

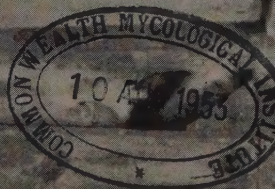
*Diseases of Stored Carrots
in New York State*

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Diseases of Stored Carrots in New York State¹

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Carrots are grown both for immediate consumption as bunching carrots and for canning. In 1943 the canning crop was produced on approximately 4000 acres in New York and brought farmers over two million dollars. In 1949 nearly a million and a half bushels of carrots were produced for canning. Most of the late crop carrots are stored for a period of time, in either common or cold storages, before being processed. It would be highly desirable if this storage period could safely be extended to 8 or 10 months, but attack on the roots during this time by a wide range of fungi and bacteria make such storage impractical. The spoilage resulting from the attacks is a definite economic factor in both production and processing of carrots and because of the losses incurred in this important crop, carrot growers, buyers, storage managers, vendors, transport men, and processors, as well as students of plant diseases, are all interested in the identities, descriptions, names, and causes of storage rots.

Losses in Storages in New York State, 1943-46

Estimates of losses from diseases in storages are very meager, except for the years 1943-46; most of the published accounts record only the sporadic outbreaks of various diseases in isolated areas. During the storage seasons of 1943-46 the United States Department of Agriculture, Emergency Plant Disease Survey, and the New York State College of Agriculture made careful surveys in the principal cold and common storages. Table 1 summarizes the estimated losses determined during these surveys of some of the storage houses in New York state.

Three important facts are not brought out in this table. First, these figures represent losses under good storage conditions, since the surveys were conducted among large storage plants with equipment for maintaining good conditions. Losses in smaller storage plants and farm storages are undoubtedly higher, because of the lack of facilities to maintain optimum conditions for storage.

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Table 1. Summary of estimated losses of carrots caused by diseases in some of the principal storages in New York state, during the storage period 1943-46

Year	Type of storage	Bushels stored	Estimated loss	
		Thousands	Per cent	Bushels
1943-44	cold common	172.0	1	1,621.5
		51.2	2	985.0
1944-45	cold common	336.6	3	8,936.9
		63.5	10	628.5
1945-46	cold common pit	257.28	3	7,341.4
		35.5	2	837.5
		4.0	7	265.0

Table 1 does not show either that when a particular lot of stored roots begins to show signs of disease it is commonly removed from the storage and sold, regardless of the market conditions. Loss in such cases may be due both to the presence of spoilage and possible poor market prices at the time of sale. Sporadic outbreaks of disease in storage, followed by removal and sale of these blocks, result in a lower total estimate of disease. This is attested to by records taken on one such lot moved from one cold storage plant on January 4, 1946. Individual scores were kept on 600 crates (1 carload) of carrots. Sixty-eight, or 11.36 per cent of the crates, were graded as badly affected; 126 crates, or 21 per cent, were graded as slightly affected. At random, 10 crates of each score class were dumped and actual counts made of the carrots with signs of disease. In the crates that had been rated as badly affected 59 per cent of the roots were still marketable, 28 per cent were completely rotted, and 13 per cent were showing signs of infection by various pathogens. In the crates rated as slightly affected 74 per cent of the roots were still in a marketable condition, 24 per cent showed signs of infection, and only 2 per cent were completely rotted. Calculations based on the actual percentages of diseased roots in each crate indicated that 6.6 per cent of the roots in this carload showed signs of infection, and 3.1 per cent were completely rotted. Thus, a total of 9.4 per cent, or 58 crates out of a single carload, could not be used by the processor.

Nor does table 1 show the extent of loss between the time of shipment from storage and that of actual processing. The figures given in table 1 represent only losses as they occur during the storage period. Stored carrots are commonly shipped to the processors in boxcars. Temperatures and humidities may rise during transit, and lots showing even a small incidence of disease at the storage plant may be valueless upon arrival at the processors. This fact has been pointed out by Lauritzen (1932), who noted high losses in transit caused by *Erwinia carotovora* and *Sclerotinia sclerotiorum*.

Field surveys made at harvest time in 1945 revealed considerable damage on the farms, particularly in the muck areas of Orleans, Genesee, and Monroe Counties. In 23 fields visited, the damage ranged from complete

loss in a number of fields to fields where damage was hardly detectable. It was noted then that the amount of damage seemed to be related to the amount of moisture to which the fields had been subjected. In one area on the West Elba muck, where the land did not have a very high water table, 3 fields of carrots that were examined showed practically no decay. On the Fancher and Clarendon mucklands, where the water was standing in the fields, the crops had completely rotted in the ground. These correlations between soil moisture and incidence of field decay were drawn from observations made at the time of the survey, and from conversations with the operators, and suggest a possible explanation for the severe outbreak of disease in the carrot fields of this region during the 1945 season.

A large number of organisms were associated with the decay found in these fields, as indicated in table 2. Having remained in the wet ground long after their normal harvest time, the roots had been subjected to continuous attacks by many organisms associated with the normal soil microflora. *Phytophthora* sp. and *Botrytis* sp. (*cinerea* type), either separately or together, were found to be the cause of most of the decay noted in this survey. *Botrytis cinerea* is commonly carried on the roots into storage, where it becomes destructive later under certain storage conditions.

The severity of the damage caused by this last pathogen was noteworthy on one farm in the Middleport muck, where 35 paces of a row (representing 500-600 roots) were pulled up at random, and all but 14 of the roots showed the grey mold rot. This same condition was found on other farms in the Shelby, Fancher, and Clarendon muck tracts.

During this survey *Sclerotinia sclerotiorum*, *Rhizopus arrhizus*, *R. nigricans*, and *Erwinia carotovora* were also commonly found to be attacking carrots. The black rot of carrots caused by *Stemphylium radicinum*, which in 1944 was common in stored carrots from the Medina region, was found only once during the surveys in the fall of 1945.

It was believed at the time of the survey that carrots harvested from fields with such a high incidence of disease would, though they might appear normal, probably carry incipient stages of decay that could develop and cause severe losses under most storage conditions. This was confirmed by taking two bushels of apparently normal carrots sorted from a field on the Shelby muck and placing them in the storage at Ithaca. The carrots were kept at 38-40°F and at 95-98 per cent relative humidity. After 3 weeks the carrots were discarded because of the large amount of rubbery brown rot (*Phytophthora spp.*) and grey mold rot (*Botrytis cinerea*) which had developed.

Table 2 lists the pathogenic organisms isolated from carrots during both field and storage surveys. Included also are the results of isolations from carrot leaves and from a large number of carrot seeds obtained from different seedsmen. Indicated in addition are the pathogenic organisms recovered from 625 isolations made from as many full and empty crates and baskets in 3 different storages, as well as the organisms found floating in

Table 2. Summary of the organisms found as causes of decay of stored carrots in New York state

Class	Pathogen	Where found				
		Root	Foliage	Seed	Crates	Air
Bacteria	<i>Erwinia carotovora</i>	+	+	0†	0	0
Phycomycetes Pythiaceae	<i>Pythium debaryanum</i>	+	0	0	0	0
	<i>Pythium ultimum</i>	+	0	0	+	0
	<i>Phytophthora cactorum</i>	+	0	0	0	0
	<i>Phytophthora megasperma</i>	+	0	0	0	0
Mucoraceae	<i>Mucor abundans</i>	+	0	0	0	+
	<i>Mucor circinellioideis</i>	+	0	0	0	0
	<i>Mucor globosus</i>	+	0	0	+	+
	<i>Mucor hiemalis</i>	+	0	0	+	+
	<i>Mucor janssenii</i>	+	0	0	0	0
	<i>Mucor mucedo</i>	+	0	0	0	0
	<i>Mucor racemosus</i>	+	0	0	0	0
	<i>Rhizopus arrhizus</i>	+	0	0	+	+
	<i>Rhizopus nigricans</i>	+	0	+	+	+
	<i>Rhizopus oryzae</i>	+	0	0	+	0
	<i>Zygorhynchus moelleri</i>	+	0	0	0	0
	<i>Zygorhynchus vuillmanii</i>	+	0	0	0	0
Ascomycetes Aspergillaceae	<i>Aspergillus niger</i>	+	0	+	+	+
	<i>Gliocladium aureum</i>	+	0	0	+	0
	<i>Penicillium expansum</i>	+	0	+	+	+
	<i>Penicillium luteum</i>	+	0	0	0	+
	<i>Penicillium oxalicum</i>	+	0	0	0	+
Ascomycetes Sclerotiniaceae	<i>Sclerotinia sclerotiorum</i>	+	+	0	+	0
	<i>Botrytis cinerea</i>	+	+	+	+	+
Basidiomycetes Clavariaceae	<i>Typhula intermedia</i>	+	0	0	0	0
	<i>Typhula umbrina</i>	+	0	0	0	0
	<i>Typhula variabilis</i>	+	0	0	+	0
Fungi imperfecti Dematiaceae	<i>Stemphylium radicinum</i>	+	+	+	0	0
	<i>Centrospora acerina</i>	+	0	0	0	0
Tuberculariaceae	<i>Fusarium roseum</i>	+	0	+	+	0
	<i>Fusarium arthrosporioides</i>	+	0	0	0	0
	<i>Fusarium vasinfectum</i>	+	0	0	0	0
Mycelia sterilia	<i>Rhizoctonia solani</i>	+	+	0	0	0
	<i>Rhizoctonia carotae</i>	+	0	0	+	0

*All plus signs in this table indicate that the organism was found on this substrate during these investigations.

†All zeros in this table indicate that the organism was not found on particular substrate.

the air in 18 three-cubic-foot samples taken in 3 rooms of one storage. From this it can be seen that the roots destined for storage are never free from organisms bent on their destruction, and that the field is the most common source of most of these pathogens. After a period of several weeks in cold storage the conidia of a number of the molds become more or less abundant, spreading the pathogens to healthy roots.

Bacterial Rots

Although Burkholder and Smith (1949) have isolated *Erwinia atroseptica* from New York carrots, the only bacterium found causing decay during the present surveys was *Erwinia carotovora* (Jones) Holland, which causes the common soft rot, also known as slimy rot of vegetables, or Jones' bacterial soft rot (Smith, 1920).

Bacterial soft rot (*Erwinia carotovora* [Jones] Holland)

This disease has been described in many countries under various names since Halsted first reported it on celery in 1891. Pammel (1895) noted what is apparently the same disease on rutabagas. L. R. Jones, in 1901, was the first to make an intensive study of its presence on carrots and, at about the same time, Potter (1899) was studying the "white rot" disease of turnips in England, subsequently shown to be caused by the same organism. Bacterial soft rot has been reported on carrots by Hirta (1927) in Japan, Park (1932) in Ceylon, Malençon and Delécluse (1937) in Morocco, Ciferri (1927) in Italy, and Strohbinder and Driabina (1935) in Russia. Many other citations could be given to emphasize the worldwide distribution of this disease.

During the present survey, the disease was found on carrots in the field and in most common and pit storages throughout the State. It was found less frequently in cold storages, presumably because the temperatures are maintained below or close to the minimum for growth of the pathogen.

Losses from and relative importance of the disease. At the beginning of the current survey it was noted that almost any lot of unwashed carrots, when placed in a moist chamber at 87°F, would be completely rotted in a few days by *E. carotovora*. Although these conditions for optimum decay by this bacterium may occur during transit, they are rarely present in carrot storages. Carrots in some cold storages showed unmistakable decay by this organism, but it was presumed to be the result of bacterial action prior to storage. Bacterial soft rot can usually be found in most common and pit storages, particularly when the storage period is extended into the spring months.

During the seasons over which the present survey extended, the winter temperatures in common storages were usually low enough to prevent excessive decay by this organism. Many common storages are equipped so that adequate ventilation is possible, thus keeping the relative humidity low enough to prohibit the establishment of the pathogen. Carrots stored in pits during warmer seasons and during the spring months become easy prey to *E. carotovora*. In one pit storage in Nassau County, more than 30 per cent of the roots were affected by this pathogen. The carrots in this pit were a total loss and, while only about 500 bushels were stored, they represented the major part of this grower's crop. Other pit storages visited during these surveys showed the presence of this bacterium, and the heavy losses it causes have been observed in the field. It is safe to conclude that few other pathogenic organisms cause a greater total loss (Chupp, 1925).

Suscepts. This bacterium has been observed attacking a wide range of vegetables, including artichoke, asparagus, brusselsprouts, carrot, cauliflower, eggplant, horse-radish, kohlrabi, muskmelon, onion, parsnip, pepper, potato, radish, rhubarb, rutabaga, salsify, tomato, and turnip (Chupp, 1925; Burkholder and Smith, 1949). This list of susceptibles could be ex-

tended to include fruits, ornamentals, and native plants with succulent parenchymatous tissues.

Symptomatology. The parenchymatous tissues attacked by this bacterium decay very rapidly, while the other susceptible tissues are usually more resistant. The cells thus attacked become water-soaked and, after losing their middle lamellae, collapse into a soft, watery, slimy mass. The epidermis usually remains intact. The rotted tissue has a greyish or brownish discoloration, which may or may not be accompanied by a foul odor, depending on the presence or absence of certain secondary organisms. The decay seems to develop most rapidly along the core of the root; when it is cut open there is usually a distinct line of demarcation between healthy and diseased tissues.

Under certain conditions of high soil temperatures and high moisture, this organism may cause a pitting of the carrot root. This has been noted in a muck tract in Wayne County, and has been reported from Richmond County on Staten Island. The field of carrots in Wayne County had been subjected to extreme conditions of high moisture during the warm September weather.

The tissues that have been partially decayed by *E. carotovora* are rapidly invaded by fungi and other bacteria. *Rhizopus arrhizus* and *R. nigricans* have been frequently found in common storages on carrots partially rotted by *E. carotovora*. In the field, *Botrytis cinerea* and *Sclerotinia sclerotiorum* often permeate these decayed tissues. Machacek (1928) has noted the frequent association on carrots of *S. sclerotiorum* with the soft rot bacillus.

These secondary invaders and "joint pathogens" make accurate diagnosis very difficult and, in certain instances, impossible.

Etiology. *Erwinia carotovora* (Jones) Holland, a relatively large and very motile, Gram-negative, bacillus with large peritrichic flagella, is the pathogen responsible for the soft rot disease. It has been characterized in a recent paper by Burkholder and Smith (1949). These investigators found that none of their isolates showed any tendency toward loss of pathogenicity. Very old cultures produced decay on carrots and potatoes as readily as did recent isolates.

The present investigations indicate that for stored carrot roots there is only one principal source of primary inoculum: the soil. Soil containing plant debris from diseased suspects of the previous year undoubtedly is the most important source. Patel (1929) noted that *E. carotovora* could live over winter in sterilized or nonsterilized loam soil held in the open at Ames, Iowa (1927-28). During December of that year the minimum temperature ranged from -25° to $+5^{\circ}$ F, and the maximum ranged from 5° to 41.5° F. The omnipresence of this bacterium on carrot roots (Luritzen, 1932) seems to indicate that the pathogen probably lives and multiplies within the soil. In the fields where this organism can sometimes be found on the aerial parts of the plant it can often be traced directly from these affected tissues down to the diseased roots.

Epiphytology. Lauritzen (1932) found a temperature of 77°F to be optimum for infection and decay and, although infection could take place at temperatures as high as 97.5°F, none occurred at 107°F, and at 36°F only an occasional infection was observed after 80 days. In uninoculated roots stored at temperatures of 32°–36°F, he observed infection by *E. carotovora* in only one out of nine seasons in lots of Danvers Half Long variety, and in one out of four seasons in lots of several other varieties. Wounding of uninoculated carrots increased the percentage of infection, but was not essential because infections were also noted in the absence of fresh wounding.

Poor ventilation during storage or transit increases the relative humidity and favors the development of decay (Johnson, 1930). Lauritzen (1932) noted that carrots generally became infected when held at a relative humidity of 90–95 per cent and a temperature of 69–77°F. The amount of disease appearing under these conditions may be influenced by its occurrence in the field; it will, however, develop to a lesser extent in the absence of soil infection. Diseased roots included in storage stock lead to a more even distribution of the disease throughout a block of carrots, and infection and spread take place over a wider range of temperatures than when the storage stock of roots is not contaminated (Lauritzen, 1932).

This bacterium is very sensitive to sunlight. Jones (1901) noted that an exposure of 10 minutes in direct sunlight killed all the bacteria on freshly poured plates; 5 minutes' exposure killed 87 per cent; 3 minutes killed 70 per cent. Long exposure to diffused light was also fatal.

Jones (1901) also noted that this organism is very sensitive to the effects of desiccation. Drops of broth cultures of *E. carotovora* on sterile slides were killed after they were dried for twenty minutes. When the cultures were diluted with water before drying, drops containing the organism were rendered sterile after 2 minutes of drying.

Control. The disease cannot always be effectively controlled in the field. The bacterium may be partly held in check by rotations with nonsusceptible crops and the elimination of diseased plant parts from the field (Chupp, 1925).

The temperature and humidity of storages are of utmost importance. Cold storages should be maintained between 30° and 32°F. Common storages should be kept as near 32°F as possible, allowing sufficient ventilation to keep the relative humidity below 90 per cent.

The carrot roots should be dried in the sun, if possible, before they are stored. Storage in crates or baskets rather than in bulk permits better air circulation, which helps to keep the surface of the roots dry.

Chupp (1925) recommends that storage houses be disinfected each season with formaldehyde (1 pint to 10 gallons of water) or copper sulfate (1 pound to 5 gallons of water). Bactericidal tests have indicated that Semesan, phenyl mercuric compounds, or even mercuric bichloride in a 1 per cent solution in water will give complete kill of this and other organisms. Care should be taken not to permit these mercury compounds

to come into contact with the roots or the storage containers, because of their toxicity to humans.

Fungal Rots of Major Importance

Cottony soft rot (*Sclerotinia sclerotiorum* [Lib.] DeBary)

Four species of *Sclerotinia*, *S. sclerotiorum* (Lib.) deBary, *S. intermedia* Ramsey, *S. minor* Jager, and *S. ricini* (now *Botryotinia ricini* [Godfrey] Whetzel) have been shown to cause decay of carrots under laboratory conditions (Ramsey, 1925). Of these species, only *S. intermedia* and *S. sclerotiorum* have been found to cause decay of carrots under storage and transit conditions, and only the latter has been noted in New York during the present study.

Four additional species can now be added to this list of diseases capable of causing decay under laboratory conditions. These new pathogens are: *S. bulborum* from onions, *S. sativa* from *Melilotus* sp., and *S. trifoliorum* from *Trifolium* sp., and *S. draytoni* from *Gladiolus* sp.

Reports in the now voluminous literature on *S. sclerotiorum* show that the disease caused by this pathogen has been found in almost every temperate and subtropical region in the world. The fungus has been found in nearly every cold and common storage visited in western New York.

Losses from and relative importance of the disease. *Sclerotinia sclerotiorum*, *Botrytis cinerea*, and *Rhizoctonia carotae* are the 3 principal pathogens affecting carrots in cold storages in New York state. *S. sclerotiorum* was responsible for great losses during the years of these surveys but, during recent years, losses caused by *R. carotae* have exceeded those caused by the cottony soft rot pathogen. Recorded losses from cottony soft rot in storages ranged from a trace to 4 per cent. Individual blocks showed a considerably higher incidence of this disease in some storages.

Carrots of carrots with only a small amount of cottony soft rot as they leave the storage may be a total loss upon reaching their destination. The higher temperatures and humidities present in freight cars during transit favor the rapid development and spread of this disease. Link and Gardner (1919), in an early report of a survey of the diseases attacking vegetables during transport, storage, and marketing, stated that cottony soft rot is the prevalent vegetable rot at low temperatures and most important rot of root crops. Lauritzen (1932) reported that this pathogen causes the greatest loss in carrots during storage and transit. Ramsey (1925) recorded a striking example of the rapidity with which this organism can attack carrots in transit. A car of carrots was harvested in Louisiana during rainy weather; they were inspected and found to be in good condition when loaded into the car. Four days later, upon arrival in Chicago, over 85 per cent of the roots in this carload were infected with *sclerotinia* rot.

This disease has been seen in epiphytotic form in the field only once. During 1945, a field of carrots on the Elba muck was completely destroyed by this organism. Aside from this one field, the disease has been found

only on scattered plants. Waterston (1938) has recorded epiphytotics of this disease on carrots growing in Bermuda.

Suscepts. The nearly omnivorous nature of this pathogen was recognized by DeBary (1886). Since his researches on this fungus, new susceptibles have been added to the ever growing list. Dickson (1930) lists a total of 172 species belonging to 118 genera and 37 families as reported susceptibles of this pathogen, with both dicotyledonous and monocotyledonous plants represented.

Coemans first reported this disease on carrots in 1860. Carrot roots have since then constituted a favorite laboratory inoculation substrate for this fungus.

Symptomatology. Lesions caused by this fungus have never been found on carrots without the presence of the characteristic cottony mycelium on the surface of the infected area. When first formed, these lesions may be mistaken for those caused by *Rhizoctonia carotae*. *Sclerotinia sclerotiorum* produces typical soft lesions on the surface of the infected root (color plate II, top). These infected areas are covered with a dense, cottony mycelium, while the lesions produced by *R. carotae* are sunken and more firm. The presence of sclerotia on the surface of the lesions confirms the presence of *S. sclerotiorum* (figure 1 b, d).

The decayed tissues are somewhat darker in color than the healthy tissues. The decay is typically a soft, watery rot (figure 1 c). It is distinguished from bacterial soft rot by the absence of sliminess. Secondary organisms frequently gain entrance into lesions caused by *S. sclerotiorum*, and quickly change the infected tissues into a soft, mushy, and sometimes slimy, malodorous mass.

Etiology. The amount of sclerotinia decay found in storages is determined by the extent of infection in the field. Healthy carrot roots placed in clean storages will remain free from this disease (Lauritzen, 1932). Infections in the storage can occasionally be traced to contaminated storage containers.

Primary cycles may be initiated at any time during the growing season. The usual source of the inoculum in nature is the soil in which the fungus is present, either as sclerotia or vegetative mycelium. Ascospores may also serve as inoculum (Dickson, 1930). The author has found that infection can also be produced by ascospores, by suspending apothecia over sliced carrots in preparation dishes. All of these were completely rotted by *S. sclerotiorum* at the end of 2 weeks. The extent to which ascospores serve as inoculum in the field is not known, but is presumed to be important. This writer observed mature apothecia in the field before carrot harvest, during the last of September (1944).

The exact method of penetration by this fungus is still debatable. Boyle (1922) believes that penetration is entirely by mechanical pressure. O'Leary (1939) tends to support DeBary's original suggestion that penetration is accomplished by the solvent action of an enzyme. Once within the suscept

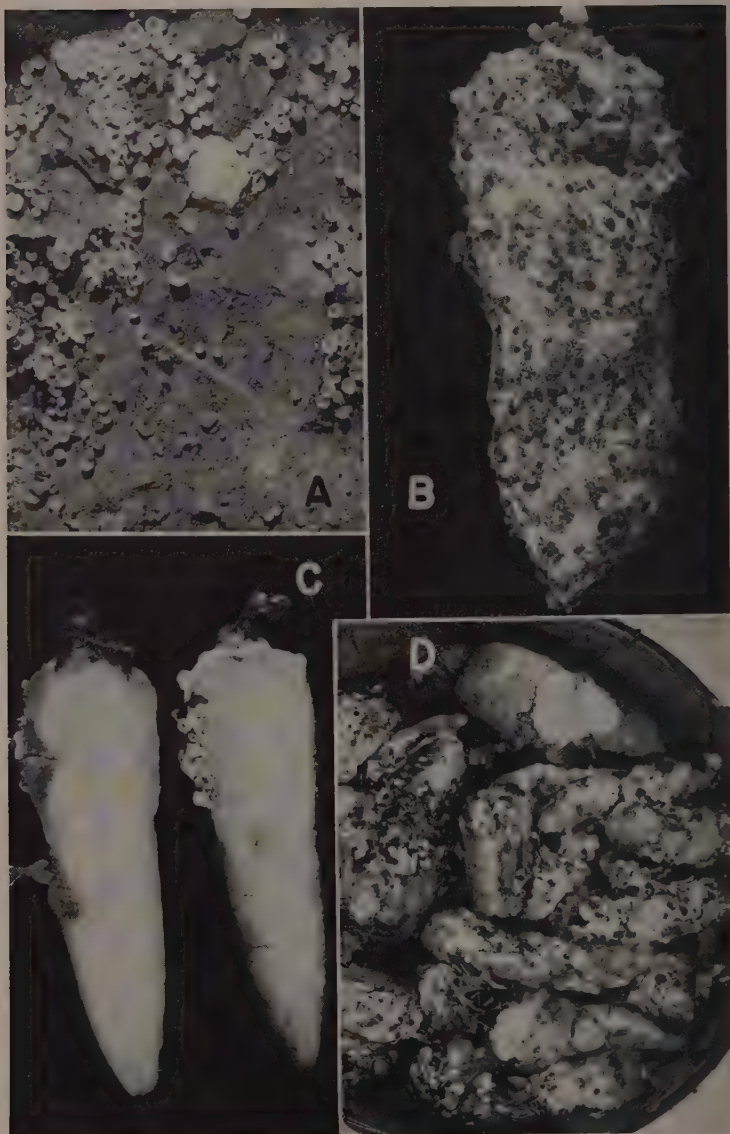


Figure 1. *Cottony soft rot*: (a) sexual stage of the organism as found in the field during spring and late fall; (b) diseased root with numerous flat sclerotia of the pathogen; (c) longitudinal section through diseased root, showing soft, watery, only slightly colored decay; (d) hamper of diseased carrots, illustrating destructiveness of this disease.

tissues, the advancing hyphae secrete a toxin which kills the cells several layers beyond the hyphal tips. Enzymes secreted by the fungus dissolve the middle lamella. The diseased tissues soon become soft and watery, held together only by the numerous hyphal threads that permeate the dead areas.

Sclerotia are formed on the cottony mycelial mat which quickly develops on the diseased roots. These sclerotia serve to disseminate the fungus and initiate other infections either by direct germination into a thallus, or by the production of apothecia and ascospores (figure 1a). Sclerotia of this fungus found in storages do not produce apothecia, since light is essential for the initiation of the perfect stage (Dickson, 1930); they have been observed, however, to initiate infections on adjacent carrots by vegetative growth.

Epiphytology. The fact that *Sclerotinia sclerotiorum* occurs only in the cool climatic zones of the world indicates the importance of temperature in its development. Dickson (1930) reported an optimum temperature for mycelial growth of 65°–77.5°F, while Ramsey (1925) determined the optimum temperature for pathogenic activity to lie between 54° and 65°F. The writer has noted growth of this organism on agar at 25°F and invasion of carrots at 32°F.

Like most other carrot pathogens, *S. sclerotiorum* needs moisture for growth and pathogenesis. Once infection has taken place, the hydrosis of the infected tissues usually supplies sufficient moisture for subsequent development and spread of the pathogen. Lauritzen (1932) noted a marked decrease in infection of inoculated roots at 80 per cent relative humidity, as compared with roots stored at 95 per cent.

Control. This pathogen is undoubtedly distributed throughout the carrot growing areas in New York state, and control measures must be directed toward keeping losses to a minimum. Since disease in the storage is generally a consequence of field infection, control measures must be undertaken there.

Beach (1922) recommended crop rotation with nonsusceptible monocotyledonous crops (for example, onions) for 2 years between susceptible crops. Other crop rotations with cereals and grasses have been suggested (Cunningham, 1927; Young and Morris, 1927). Crops to be avoided include lettuce, celery, beans, the cucurbits, and late cabbage. For muck growers potatoes and onions are among the best to grow. In some vegetable growing areas, Cyanamid has been disced thoroughly into the top 3 or 4 inches of the soil at rates of 700 to 1200 pounds per acre, 21 to 36 days before planting, with good results.

Control in storage is limited to the elimination of the pathogen from storage containers and maintenance of proper conditions of temperature and moisture within the storage house.

It is recommended that containers to be used for storing carrots be placed outside in a dry place exposed to the sun during the period of

the year in which they are not in use. This procedure hastens the deterioration of untreated crates but, in many instances, will more than repay for this loss in the prevention of carrot decay. In certain instances, dipping in chemical wood preservatives has been recommended. This procedure may serve the twofold purpose of killing contaminating organisms and preserving the crates from decay by wood-destroying fungi, but the odor of some wood preservatives may be lasting and deleterious to carrots held in closed rooms.

The maintenance of a temperature near freezing and a relative humidity near 85 per cent will check the development and spread of this fungus in the storage houses. Storage in crates or hampers rather than in bulk is recommended, because temperatures and humidities are kept lower within the pile and the space between the crates retards the spread of the organism.

Grey mold rot (*Botrytis cinerea* Pers. = *Botryotinia fuckeliana* [DeBary] Whetzel)

The organism *Botrytis cinerea* is worldwide in distribution. Records in the literature indicate that this fungus has been found in 43 countries, representing every continent of the world and the islands of the Atlantic and Pacific Oceans.

There are, however, relatively few records to show that this fungus attacks stored carrots. This is surprising, because it is common in storages and always causes some damage to roots stored two or three months or longer at temperatures between 32° and 43°F. During 1944-46, *B. cinerea* was found in every cold and common storage visited. It has also been found in fields of carrots in Orleans, Genesee, and Monroe Counties.

Losses from and relative importance of the disease. As a general rule, losses caused by *Botrytis* are not very great, except in blocks of carrots stored for two to three months or longer. During the present study, losses have ranged from a trace in most storages to 2.25 per cent in storages where the humidity was near saturation.

This organism spreads readily in the storage, either vegetatively from one carrot to another or by air-borne conidia. It also invades lesions caused by other less virulent fungi. It thus remains a powerful threat to carrots intended for storage over extended periods of time.

B. cinerea rarely has been recorded as causing decay of plants growing in the field. During the 1945 season, however, severe losses caused by this pathogen were noted in the mucklands of Monroe, Genesee, and Orleans Counties. This epiphytotic was probably caused by the very wet ground the carrots were left in long after their normal harvest time; heavy rains prevented growers from pulling the roots when they were mature.

Suscept. Corresponding to its nearly ubiquitous distribution, *B. cinerea* is somewhat omnivorous in its susceptible range; it is recorded as pathogenic on 154 species of plants. These represent such diverse groups as fern, fig,

guava, hemp, larch, cactus, pine, elm, lily, cereals, and asparagus. A strain of this fungus has been recorded as being parasitic on man in England and in Hungary. There are numerous records that show the isolation of this fungus from the soil (Gilman and Abbott, 1927).

Laboratory inoculations with four isolates of *B. cinerea*, representing different cultural variants, produced decay on potato, parsnip, rutabaga, beet, and cabbage. In the same way, infection was produced in carrot roots inoculated with cultures of this organism isolated from celery, cabbage, blackberry, rose, dandelion, geranium, bean, beet, begonia, tulip, dodder, lettuce, pansy, and peony. Although pathogenic races of this fungus are known (Paul, 1929), these inoculation experiments seem to indicate that only one strain was found to be present in New York State on certain vegetables and ornamentals. In addition to cultures of *B. cinerea* (*Botryotinia fuckeliana* [DeBary] Whetzel), the writer had access to a number of additional species of *Botrytis* from the culture collection of the late H. H. Whetzel. Mature carrot roots (variety Danvers Half Long) were inoculated with these and incubated in moist chambers at 43°F for 3 weeks. The following species are pathogenic on carrot roots and represent potential storage pathogens: *Botrytis convoluta* on *Iris* sp.; *B. allii* and *B. squamosa* on *Allium cepa*; *B. iridis* on *Iris* sp.; and *B. symplocarpeae* on *Symplocarpus foetidus*.

Symptomatology. Lesions caused by this pathogen may occur anywhere on the root, although they are found most commonly either on the crown or on the tip (figure 2b and color plate I, bottom). The infected tissues are light brown in color and water-soaked at first. The affected areas later appear somewhat spongy. The diseased cells do not separate; thus, the affected tissues are almost leathery in the advanced stages of decay. Once seen, this decay is distinct enough to make diagnosis fairly easy (figure 2c).

The surfaces of the lesions usually become covered with the characteristic greyish-brown conidiophores and conidia and, in some cases, are accompanied by blackish sclerotia closely appressed to the decayed tissues (figure 2a). At temperatures near freezing the aerial hyphae on diseased roots are frequently white, and the conidia may be absent. The presence of sclerotia and/or the characteristic decay of the susceptible tissues serve to identify the disease in these instances.

Etiology. The appearance of this organism in almost every storage in New York State indicates a widely distributed source of inoculum. The presence of *B. cinerea* in the soil was indicated by its appearance on carrots in the field over a wide area in western New York during the 1945 season. This fungus has been isolated from the soil in New York (Jensen, 1912). It is believed that the organism growing on debris in the soil constitutes the principal source of primary inoculum.

Infection may take place in the field during periods of cold, wet weather. In 1945, carrots subjected to such conditions before harvest showed lesions caused by this organism as they went into storage. The



Figure 2. Botrytis, or grey mold rot: (a) three expressions of the disease commonly found in storages: brownish lesions without any evidence of fruiting structures of pathogen, lesions covered with conidia, and characteristically contorted sclerotia of pathogen; (b) beginning infections on root; (c) longitudinal section through diseased root showing characteristic decay.

presence of this disease in the field, however, is not a necessary condition for its later appearance in storage. Lots of carrots which appeared perfectly normal at harvest time showed 1-2 per cent grey mold rot after three months' storage. It was in all likelihood carried into the storage either with the soil particles clinging to the roots or as an incipient infection.

In 2 to 4 weeks, at temperatures maintained in cold and most common storages, typical grey mold rot lesions appear on the roots. The decay progresses fairly rapidly throughout the tissues, so that in another 2 to 4 weeks the root is completely decayed. During this period the pathogen exhibits itself as a mat of greyish-brown hyphae which may bear numerous conidia and/or blackish, flattened, contorted sclerotia closely appressed to the decayed tissues.

Freehand sections made through the edges of the lesions showed this fungus to be both inter- and intracellular. Blackman (1925), working with *Botrytis cinerea* on the broad bean, noted that the cells of the susceptible were killed in advance of the hyphae by the secretion of an enzyme of the pectinase type, and the dead tissues served as food for the pathogen. These dead cells are readily penetrated by the actively growing hyphae.

Secondary cycles of the disease in storage may be initiated by air-borne conidia, 7.2 per cent of the spore population of the air having been found to be of this organism. New cycles may also result from vegetative hyphae growing from carrot to carrot, and from the storage containers to the roots. In fact, more than 11 per cent of the organisms recovered in some 625 isolations from crates and baskets were *B. cinerea*. Crates may thus be considered as sources of primary inoculum at the beginning of the storage season.

The development of the disease in secondary cycles follows the same pattern as that of the primary cycle, and, because of the overlapping of the two cycles, they are soon indistinguishable.

Epiphytology. Pathogenesis by *B. cinerea* is critically dependent upon several external conditions, of which temperature, moisture, and an external source of nutrients before penetration are the most important.

Spores have been observed to germinate at 32°F after 18 days. The fungus has been observed to grow on agar at temperatures from 29° to 92°F, with an optimum at about 76°F. This agrees closely with the figures given by Lauritzen (1932). Infection and development of the disease take place, however, over a somewhat more restricted temperature range: the widest range of temperatures observed was from 29° to 77°F. The maximum amount of decay was obtained at 73°F, which is very close to the temperature for optimum growth of the fungus on agar. Lauritzen noted a drop in the percentage of infection as the relative humidity fell from 90 to 70 per cent. There was no infection on roots held at 70 per cent, either on uninoculated checks or those inoculated and dried before incubation, and inoculated roots stored wet developed only 2 per cent infection at this relative humidity.

In infection studies with this pathogen it was found that washed roots dipped in a spore suspension of *B. cinerea*, or inoculated by the "well"-method³ and subsequently incubated in moist chambers at 43°F, seldom became infected. If, however, the spores were suspended in a 4 per cent solution of peptone, ammonium sulphate, or potassium nitrate, infection readily took place. Spores washed in glass distilled water and centrifuged twice failed to germinate without the addition of nutrients.

Vasudeva (1930) noted that *Botrytis allii* could be made to parasitize apple tissue by adding to the inoculum certain nitrogenous substances, such as asparagin, ammonium nitrate, potassium nitrate, or peptone. The effect of these nitrogenous compounds in enhancing infection was found to run parallel with their effect in stimulating the secretion of pectinase. Lauritzen (1932) has also noted that infection of carrots by this fungus is aided by the presence of some nutrient medium.

Under storage conditions supplemental nutrients are almost universally present, either in the form of soil solutes on the root, senescent or dead tissues in the tops, side roots, wounded areas, or in the fine tip ends of the tap root. Virgin (1942) noted that most grey mold decay developed from the crown; the end of the tap root was next in vulnerability.

B. cinerea is apparently unable to penetrate the unbroken epidermis of the carrot root. In an experiment by the writer, 10 carrot roots were inoculated by placing a small bit of mycelium on what appeared to be an uninjured area. At the end of 3 weeks' incubation at 43°F only one of these inoculations was successful. There was 100 per cent infection when the roots were cut or scraped with a scalpel prior to inoculation.

Control. Care in harvesting and handling roots before storage, in order to reduce the amount of mechanical injury, may do much to reduce the amount of grey mold rot in the storage. A very considerable amount of bruising results from the stacking of crates on the field trucks, their subsequent transfer to larger trucks which take them to the storage, their reloading on to push carts, and, finally, their stacking in the storage room. Care not to fill the crates too full in the field is the first step in reducing the number of crushed, broken, and bruised carrots, and the number of points where infection can take place.

In actual storage practice the control of temperature and humidity are the principal means now employed to reduce the amount of decay caused by this fungus. At a temperature near 32°F the organism develops very slowly. Lowered humidities also serve to check the development of this disease, but humidities low enough to check it completely will cause excessive shrinkage.

Blocks of carrots usually cannot be stored for extended periods of time because of this disease. Some more effective method of control must be resorted to in order to keep carrots longer than 4 to 5 months in most

³Method consists of removing a small shallow cylinder of tissue from the carrot root with a cork borer. The inoculum is placed at the bottom of this well and the cylinder of tissue replaced in the hole.

storages. All drip from the cooling coils and ceiling onto the carrots should be prevented, and every possible effort made to reduce the temperature of the crop to 31 or 32°F as soon as it is brought to the storage room. Defrosting one coil at a time, rather than a whole bank of coils or an entire room, serves best to avoid elevation of the air temperature and the resulting sweating of the roots in rooms where the humidity is very high. Wider use of forced air circulation, as in modern apple storages, would probably help keep dangerous surface films of moisture down to a minimum.

Crater rot (*Rhizoctonia carotae* Rader)

Since this new disease has reached epiphytotic proportions in the storages of western New York and Illinois, it is considered one of the serious carrot storage rots (Rader, 1948).

Ramsey (1934) first called attention to it on receipt of specimens shipped to him by one of the state inspectors at Rochester, New York. During the New York surveys made in 1943-46, this pathogen was found on carrots in cold storages in Orleans, Monroe, and Wayne Counties. In one storage, heavy losses were sustained in upland grown carrots. Ramsey (1949) has also noted severe losses caused by this disease in Illinois-grown carrots.

Losses from and relative importance of the disease. Crater rot may reach epiphytotic proportions and cause heavy losses in cold storages where the relative humidities are high. Because the causal organism can flourish at the temperatures maintained in cold storages, its presence has caused heavy losses. The disease was first brought to the attention of the staff at the Cornell Agricultural Experiment Station by the Fairport Storage Co., in 1942-43. It had been noted previously, but during that season there were severe losses at several cold-storage plants. During the next two storage periods (1943-44, 1944-45) losses from this organism were light. However, in 1945-46 the disease was found in epiphytotic form in several cold storages; in one of them an estimated 10 per cent of the roots developed lesions by January 1. In a second cold storage, an average of 4 per cent of the roots were affected, with certain blocks running considerably higher. One block of carrots at a cold storage in Williamson, New York, showed an estimated 5 per cent of the roots affected.

Ramsey (1949) reported that one of the large soup companies in Chicago stored about 22 million pounds of carrots in several cold storages during 1948-1949. Crater rot began to appear in these carrots during February, and lots of carrots removed during March showed from 5 to 90 per cent infection. The most severe losses were in the top layers of baskets stored in very humid rooms. The company reported that their loss on this lot of carrots ran well over \$100,000. Large numbers of baskets were so badly decayed that none of the carrots could be processed. Extra labor, amounting to about \$5000 a week during the canning season, was required to peel and pare out the moderately affected roots.

Suscepts. Inoculations with this pathogen into celery, potato, beet, parsnip, and rutabaga have given negative results. A white-colonied, flocculent, clamp-forming organism was isolated from parsnips⁴ on which it was found to cause a brownish rot similar to that produced by *Pellicularia filamentosa* (*Rhizoctonia solani*). This clamp-forming fungus appeared very similar in culture to *R. carotae*; however, inoculations into both carrots and parsnips gave negative results. The known susceptible range of *Rhizoctonia carotae* is thus far limited to carrot roots.

Symptomatology. Crater rot appears on the roots first as small, whitish hyphal knots. Small pits then appear under these hyphal knots. At this stage the disease can readily be confused with the fusarium dry rot disease of carrots. The pits enlarge into sunken craters lined with a white, flocculent mycelium (figure 3 and color plate I, top). Because of these characteristic craters, the name "crater rot" has been given this new disease (Rader, 1948). In storages with high humidities the fungus mycelium spreads rapidly from these craters, until entire crates are covered with the white, cottony mycelium (figure 4a and front cover). The disease at this stage has an appearance similar to that of the rot caused by *Sclerotinia sclerotiorum* (which often invades these tissues), but it is distinguishable from it by a looser, web-like mycelium and the absence of sclerotia. An abundance of clamp-connections on the hyphae confirms the presence of this fungus (figure 4b).

Botrytis cinerea and *Sclerotinia sclerotiorum* are found commonly as secondary invaders in these lesions (figure 4c,d). The characteristic symptoms of crater rot may thus soon become obliterated by these more rapid-growing fungi.

Etiology. Little is known about the life history of this pathogen. The fungus may follow the same developmental pattern as other species of *Rhizoctonia*. It is presumably a soil inhabitant. The disease is probably initiated before harvest time, or shortly after the roots enter storage, for no visible symptoms have been found at harvest time on lots of carrots which later develop it. Carrots grown on land previously planted with other crops for several years showed the disease subsequently in storage. The fact that the disease is not evident until after 3-4 weeks of cold storage does not necessarily indicate that infection takes place after the roots are stored; incipient infections may occur in the field.

Storage containers may also serve as sources of primary inoculum for this disease. In one storage, 19.5 per cent of more than 600 isolations made from crates and baskets yielded this fungus. Direct microscopic examination also revealed its presence on storage containers.

Infection may take place anywhere on the fleshy root; the spotted appearance of the lesions on the roots suggests penetration through areas left by dead side shoots.

The initial disease symptoms develop slowly, and are often not evident before 1 to 2 months of cold storage. Once they appear, however, the

⁴Subsequently determined by R. E. Wilkinson (1952) to be an *Itersonilia* sp.



Figure 3. Crater rot: typical craters produced during advanced stages of the disease.

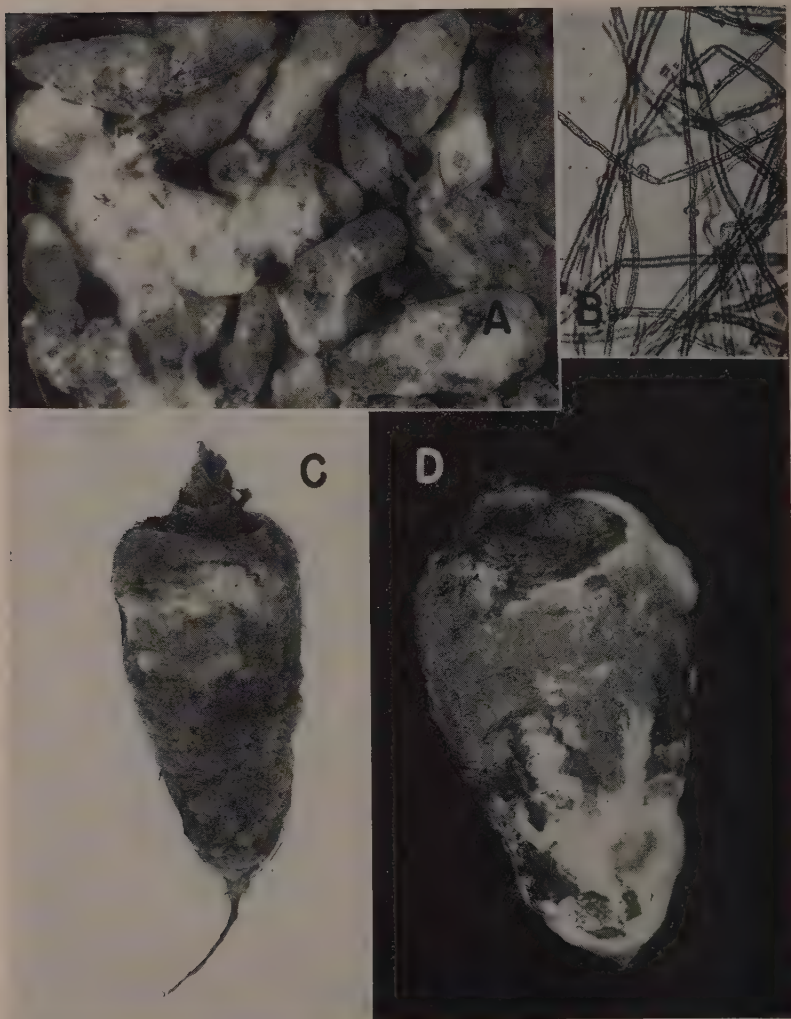


Figure 4. Crater rot: (a) crate of carrots showing advanced stages of decay; (b) photomicrograph of hyphae of *Rhizoctonia carotae* showing clamp connections; (c) crater rot lesion invaded secondarily by *Botrytis cinerea*; (d) crater rot lesion invaded secondarily by *Sclerotinia sclerotiorum*.

disease seems to develop fairly rapidly; a period of 2 to 3 weeks at 32°F is sometimes all that is required to render a root worthless for processing.

The pathogen produces an abundance of mycelium over the affected areas; adjacent roots are soon covered with it and fall ready prey to its attack. This fungus grows along the wooden slats of the container, spreading to other crates of carrots. A series of secondary cycles are initiated by its rapidly growing mycelium.

Epiphytology. The difficulty experienced in demonstrating the several expressions of this disease in the laboratory indicates rather narrow temperature and moisture requirements for its infection and development. Observations in storages bear this out. The greatest incidence of this disease was noted in the two storages where the humidities were maintained at nearly 100 per cent. The disease has been less frequent in common storages or in cold storages in which, by reason of construction or management, the relative humidity was held well below saturation.

The pitting type of symptom has been produced in the laboratory by wrapping unwashed inoculated carrots in wet paper toweling and placing them in a moist chamber at temperatures between 32° and 41.5°F. The "well" method of inoculation usually produces the typical soft rot associated with the pathogen.

It has been found that on agar this organism grows over a temperature range of from 25° to 76°F, with the optimum at 70°F. Infection has been obtained from 32° to 59.5°F on carrots artificially inoculated and placed in moist chambers.

Control. Since the primary source of inoculum for this disease is believed to be in the soil, rotation with nonsusceptible crops would appear to be the best way of lowering the amount of inoculum in the soil. However, the appearance of the organism in carrots grown on land not previously used for carrots, or which had been planted to other crops for a number of years, and the limited knowledge of the suscept range of this pathogen discourage such a recommendation at present.

Infection in the laboratory is dependent upon high humidity. It follows, then, that similar conditions are necessary for infection in the field or storage. Any cultural practice to keep the top soil moisture low — such as clean cultivation, elimination of weeds and a wider spacing of the plants in the moist portions of the field — might lower the incidence of field infection.

One of the chief difficulties in many cold storages is an inability to bring the temperature of the carrots down to 32°F promptly enough as they are received in the fall. This might be done with better air circulation, provided the rooms are not overloaded. Proper storage management should keep humidities low enough to prevent excessive spread by this pathogen.

Infested storage containers are also a source of primary inoculum of this disease. When crates are not in use they should be placed in the open.

Crates that have been used to store lots of carrots with this disease the previous season should be treated with a suitable chemical solution.

Licorice rot (*Centrospora acerina* [Hartig] Newhall)

Although known for more than half a century on the European continent, licorice rot has been recognized for only a comparatively few years in North America. *Centrospora acerina* was first detected in New York on celery (Newhall, 1944). It is common on stored celery in western New York and in the Ontario region of Canada. The pathogen was first recorded on stored carrots in New York during 1945 (Rader, 1945). It is known in Europe from Denmark, Germany, and France. Newhall (1946) studied the taxonomy of this fungus and established its identity with the European organism.

Losses from and relative importance of the disease. On celery, licorice rot, or black crown rot as it is known on this suspect, has caused considerable losses in certain areas. Truscott (1944) states that in Ontario by the end of the cold-storage period in February, 20 to 100 per cent of the stalks may be rendered worthless by this pathogen.

Individual lots from infested carrot fields have developed more than 30 per cent of licorice rot after a 4-month storage in Ithaca at 61°F. Some blocks of carrots have been found in cold storages with from 1 to 2 per cent of the roots affected but, in general, losses have not been more than a trace.

Suscepts. Hartig (1880) originally described the fungus on *Acer* seedlings. Osterwalder (1924) described what is now believed to be the same fungus on *Viola tricolor*. Westerdijk and Van Luijk (1924) added *Carum carvi* to the suspect list. Neergaard (1942) described this fungus as occurring on parsley roots. Newhall (1944) recorded celery as a suspect, and noted that decay was produced on inoculated apple and carrot. The latter suspect was found infected naturally in the field by the writer (1945). Tompkins and Hansen (1950) described heavy losses to pansies in California and they reported a number of ornamental bedding plants as hosts. Neergaard (1945) has reported the fungus on *Primula malacoides*, and Neergaard and Newhall (1951) have brought the list of known suspects to 47.

Symptomatology. Lesions caused by this fungus may occur anywhere on the carrot root. The affected tissues are watersoaked and brownish at first, soon turning a charcoal black. The advancing margin of the lesions always retains this brownish, watersoaked appearance (figure 5). This characteristic margin of the lesions distinguishes the disease from the black rot of carrots (*Stemphylium radicinum*), which produces lesions with discrete, black margins. The affected tissues in licorice rot are very soft and, under conditions of high humidity, somewhat watery. Diseased roots from drier storages show a somewhat less moist, more punky type of decay.



Figure 5. *Licorice rot*: longitudinal section through diseased roots, showing diffuse margins of lesions.

Etiology. Actually, very little is known concerning the life history of *Centrospora acerina*. Investigations during the present study indicate that the fungus can live in the soil in the absence of susceptibles for at least ten months. This permits the organism to overwinter in the soil, a fact recently substantiated by direct isolation of the fungus from Wayne County, New York, muckland soil by Neergaard and Newhall (1951).

Apparently infection can take place any time during the life of the suspect. Mature plants have been found in the fields with lesions caused by this pathogen. More commonly, symptoms appear on roots several weeks after they are placed in storage.

The mode of infection is not known. It presumably follows the same pattern as that of related fungi; that is, penetration by mechanical pressure of the infection hyphae. Cross-sections made through lesions show cell disorganization and plasmolysis in advance of the invading hyphae. The mycelium within the suspect is intercellular.

Conidia have never been observed on lesions on carrot roots; they may possibly be produced under conditions of very high humidity, as they are on celery, and may serve to disseminate the fungus. Vegetative growth of the hyphae has also been observed to spread from one root to another.

Epiphytology. *Centrospora acerina* grows well in pure culture over a temperature range of from 32°F to 76°F, with a maximum growth near 65°F. Infection on carrots in the laboratory has been obtained from 32°

to 70°F. The greatest spread through the tissues has been obtained at 60°F. Infection can take place through the intact epidermis. Moisture is essential for the penetration of the organism. Inoculated roots placed in moist chambers without added water seldom become infected, but infection always takes place when water is added to the bottom of the moist chambers in which the inoculated carrots have been placed. This indicated the necessity of a very high humidity for infection.

Control. The present practice of maintaining temperatures near freezing and a humidity down to 90 per cent may help reduce spread of this disease, but will do little to prevent infection of the roots as they come out of the ground, or subsequent development of the disease in infected roots in storage. The avoidance of celery in the rotation, and the prevention of all drip on stored carrots may be helpful in keeping losses down.

Black rot (*Stemphylium radicinum* [M., D. & E.] Neergaard)

This disease was first discovered in America during the winter of 1918-19 by Meier, Drechsler, and Eddy, in the New York markets and on Long Island. Examination of carrots from Danvers and South Peabody, Massachusetts, revealed the presence of the disease in these localities (Meier, Drechsler, and Eddy, 1922).

Neergaard (1937) presented evidence that this disease had been long known in Denmark, but was confused with several other carrot troubles. This author indicated that E. Rostrup found a *Macrosporium* sp. pathogenic in 1887 on the roots of carrots; he referred to it as *Macrosporium dauci*. In describing the disease caused by it he stated, "...it appeared on the leaves, which assumed a black color down to the neck of the roots." He repeatedly referred to this disease in his surveys of the maladies of agricultural plants in 1893, 1899, 1902, 1903, and 1904. In 1902, Rostrup (Neergaard, 1937) added to his description as follows:

M. dauci (Kühn) not infrequently appears in August on tops of carrots as blackish-gray spots on the outermost leaves, which at last become quite black, dry and rolled up. Gradually it spreads to all the leaves...so that the whole of the top becomes black...on through the petioles down to the upper part of the root, the whole top of the root becoming black and interwoven with hyphae. Sometimes the disease spreads in such manner that large spots in the field appear to be attacked.

Neergaard has carefully examined Rostrup's original preparations (at the Phytopathological Laboratory of the Veterinary and Agricultural College, Copenhagen) from which the above diagnosis was made, and has satisfied himself that Rostrup was actually working with *Stemphylium radicinum*. Rostrup, then, in 1887, was the first to observe what is now known as *S. radicinum* on carrot, without realizing that it was a new species.

This seed-borne disease of carrots is widely distributed, having been reported from Italy (Scaramella, 1929), Canada (Mounce and Bosher, 1943), England (Salmon and Ware, 1934), Bulgaria (Christoff and Cristova, 1939), Russia (Gutner and Khokhrjakov, 1940), Germany (Bolle, 1924), and Sweden and Hungary (Neergaard, 1945). Within the United States,

it is known in New York (including Long Island), Massachusetts, Pennsylvania, Illinois, and Idaho. During the present survey it was found in nearly all the storages visited.

Losses from and relative importance of the disease. It is difficult to assess accurately the losses caused by *S. radicum*, since the lesions on stored roots produced primarily by this pathogen are readily invaded and masked by other fungi such as *Botrytis cinerea* and *Sclerotinia sclerotiorum*.

The most conspicuous losses are from the lesions produced on the roots of the stored carrots. This pathogen causes considerable decay, besides providing wounds through which more active decaying organisms may gain entrance.

The leaf and petiole lesions, described on carrots on the European continent (Bolle, 1924), apparently do not occur as commonly in the United States.

Lauritzen (1926) gives the following loss figures for carrots stored for 185 days at 32°–36°F: 1920–21, 58 per cent; 1921–22, 2.4 per cent; 1922–23, 13.2 per cent. In a carlot of carrots shipped from Williamson, New York, 62 per cent of the roots developed black rot during the 185-day storage period at 32° to 36°F.

Neergaard (1937) and Bolle (1924) describe the pathogen as causing a damping-off or "blight" of the seedlings. Mounce and Bosher (1943) record this same type of symptom on carrot seedlings in North America. Seedling blight, which results from planting infested seed or planting into infested soil, can materially reduce the stand in a field.

Suscepts. Several additional susceptibles have been found since this disease was first described on carrot roots. Neergaard (1932) listed, in addition to carrot seedlings, those of celery, parsnip, and parsley as susceptibles and, later (1945), the seeds of *Linum usitatissimum* and a *Malva* sp.; Groves and Skolko (1944) have also isolated this fungus from the seeds of *Cucurbita pepo*.

Symptomatology. The lesions produced by *Stemphylium radicum* may occur on any portion of the root. On the crown, the rot generally penetrates rather deeply along the core (figure 6a,b and color plate II, bottom). The lesions on the sides of the roots are typically circular in outline, shallow, and slightly depressed. On the tip of the roots the rot may involve most of the lower portion. It is black in color and has no odor. In moist storages it appears as a soft, wet rot; roots from a dry storage room have a more characteristically dry, mealy type of decay (Lauritzen, 1926; Neergaard, 1937). The lesions are characterized by the discrete, black margins which separate the diseased from the healthy tissues.

On seedlings, both root and hypocotyl may turn black while the leaves remain green. The seedling dies as the water supply is cut off by the decaying tissues (Neergaard, 1937). The disease appears on the leaves and petioles of the plant as small discolored areas, at first brown, then turning black. On the petiole these spots often extend into the vascular tissues,

causing the entire leaf to wilt and die (Meier, Drechsler, and Eddy, 1922). On second-year plants, grown for seed, the disease appears as spots along the seed stalk and in wet weather causes a decay of the inflorescence. The stalk may decay, so that the umbels wither before setting seed (Bolle, 1924), and the seeds may become infested in the less affected umbels (Neergaard, 1937).

On diseased roots and stems exposed to high humidity, the fungus grows on the surface of the lesion, producing numerous black conidia. The mycelium formed in the lesions on the roots sometimes has a greenish cast (Neergaard, 1937).

Etiology. This pathogen attacks carrot seedlings, foliage on the maturing plants, and roots in the field and in storage. Infections on the roots may develop through wounds, decaying rootlet bases, or through the unbroken epidermis (Lauritzen, 1932).

Scaramella (1929) states that the fungus can grow in the soil. Pots of soil infested with this pathogen were placed outside during the winter of 1946 at Ithaca, New York. Carrot slices placed in these pots the following spring soon developed black decay. *S. radicinum* was isolated from these pieces of carrots. It is quite probable that once established in the soil, this fungus regularly survives the winters in New York state.

The disease is readily spread to new areas by infested seeds. Diseased plants growing from these seeds are generally killed; the fungus then spreads through the soil, initiating secondary cycles on adjacent plants or saprogenic cycles in the soil.

In storage, the disease can be spread by vegetative growth of the fungus from one root to another. Spores are formed on the diseased areas on roots and, undoubtedly, serve to spread the fungus in storage. Spores of this pathogen were not, however, recovered from 18 samples of air taken in one storage.

Epiphytology. While infection and decay can take place slowly at the minimum temperature of 31°F for growth of the pathogen, maximum growth and decay occur at 83°F, and Lauritzen (1926) found the fungus unable to grow at temperatures above 103°F.

Neergaard (1937) stated that the disease spread rapidly on leaves and petioles that were maintained at a high humidity (95–98 per cent). The attacks were reduced when plants were kept at relative humidities of 60–75 per cent. In the laboratory, infection on roots is dependent upon high humidities.

Control. The seed-borne nature of this fungus has been established by Neergaard (1927); Mounce and Bosher (1943); Groves and Skolko (1944). Seed treatment can prevent the entrance of this fungus into uninvaded areas. Doyer (1927) found that *S. radicinum* could be eliminated from the seed by treating it for one hour with ¼ per cent Germisan or ¼ per cent Uspulum solutions. Neergaard (1937) confirmed the work of these two authors, adding Sanagran (¼ per cent) to the list of seed disinfestants; he

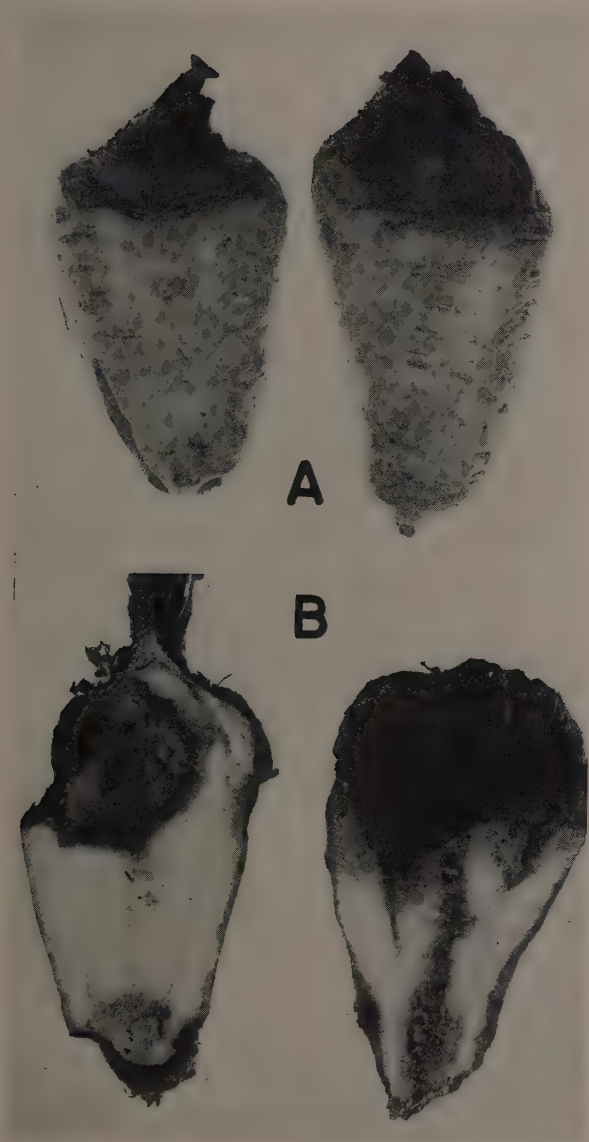


Figure 6. Black rot: (a) crown rot caused by *Stemphylium radicinum*; (b) comparison of black rot (left) lesion, characterized by discrete margins, and licorice rot (right), with margins of lesions brownish and indistinct.

also noted that red copper oxide gave little control of this organism. Mounce and Bosher (1943) obtained good control of this disease by treating the seeds with the following dusts: Semesan Junior (1 per cent ethyl mercury phosphate), New Improved Ceresan (diluted to 1 per cent ethyl mercury phosphate), and Spergon (tetrachloroparabenzoquinone).

In storage, temperatures near freezing retard the development of this disease. Maintenance of relative humidities below 90 per cent of saturation also improve control.

Fungal Rots of Secondary Importance

Woolly soft rot (*Rhizopus arrhizus* Fisch., *R. nigricans* Ehrenb., and *R. oryzae* W. & Geer.)

This disease is common on both bunched and topped carrots in transit or in markets. It is frequently serious in common storages but never in cold storages. Three species of *Rhizopus* (*R. arrhizus*, *R. nigricans*, and *R. oryzae*) have been found associated with woolly soft rot in New York. *R. arrhizus* is more widespread in storages and, on the basis of inoculation tests, is a more virulent pathogen than the other two species. All of them are common inhabitants of the soil and are widely distributed throughout the world (Gilman and Abbott, 1927).

When carrots are placed in moist chambers at 81–87°F, rhizopus rot will develop along with bacterial soft rot on most samples of unwashed carrots grown in New York State, but the disease rarely causes much loss at the temperatures maintained in cold storages. However, the presence of the spores of the fungus on almost all lots of carrots should caution processors and distributors against delays in handling the roots once they have been taken from the storage.

Suscept. Species of *Rhizopus* commonly occur on a wide range of vegetables and fruits. These species can also flourish on a great variety of decaying vegetable debris and can live in the soil.

Symptomatology. The decay may begin anywhere on the root, although the crown and mechanical injuries on the root constitute a common point of entry for this pathogen. The affected tissues are watersoaked and slightly brownish in color. The decay is firmer than that caused by the soft rot bacterial. The presence of the hyphal threads in the diseased tissues can be easily demonstrated. The coarse white mycelium of the fungus is formed abundantly over the old lesions.

Etiology. The life history of one of these fungi in culture has been carefully investigated by Blakeslee (1904), but their developmental cycle in nature is less fully known. The organism growing in the soil undoubtedly constitutes the greatest source of primary inoculum for this disease. It is probably carried into the storage either with particles of dirt clinging to the roots or as young thalli established in the dead petioles on the crown. The decay develops very rapidly at high temperatures; two days at 87°F are enough to destroy a root completely. At lower temperatures decay takes longer.

Secondary cycles develop rapidly under the proper conditions of moisture and temperature. The pathogen is spread readily by air-borne sporangiospores (table 2, p. 6), or by vegetative hyphae growing from one root to another.

The zygospores formed by the sexual union of compatible strains may play a part in sustaining the fungus under adverse conditions, such as dry periods in the summer and during the time of storage.

Epiphytology. The attacks by these pathogens are limited to carrots that have been subjected to unusually high temperatures. Infection rarely takes place below 69° F, although Lauritzen (1932) found that it could occur as low as 36° F. The optimum lies between 87° and 98° F, with a maximum somewhat higher.

Control. The pathogens are readily controlled in cold storages, because the temperatures are well below the minimum for infection and growth of these fungi. During most seasons temperatures, even in common storages, usually are low enough to prevent any serious losses. Adequate ventilation in common storages will keep losses to a minimum during the warm portions of the storage season. This disease presents a problem in shipping carrots, and serious losses can usually be expected in lots where the temperature has been permitted to rise above 83° F.

Fusarium dry rot (*Fusarium roseum* Lk. ex. Fr. emend. Snyder and Hansen)

There are very few published records of an attack of *F. roseum* (*F. avenaceum*) on carrots. These meager accounts indicate its occurrence in Denmark (Neergaard, 1937) and Germany (Wollenweber and Reinking, 1935) and give only a brief characterization of the disease. This is presumably the same disease studied by Lauritzen (1932) under the name of fusarium rot.

The disease is found commonly in New York storages, though losses during the current surveys were very minor.

Suscepts. *Fusarium roseum* has been recorded on a great number of susceptibles. It occurs in the United States and England as a root rot of cereals (Bennett, 1928) and potatoes (McLean and Walker, 1938; Moore, 1945). Wollenweber and Reinking (1935) state that this fungus is distributed widely in the temperate zone and has been found on more than 150 plant species, including grasses and cereals, truck crops (potatoes, beets, etc.), legumes, deciduous and evergreen trees, fruit trees, shrubs, lichens and fungi (species of *Exobasidium*, *Scleroderma*, *Claviceps*, and others), dead beetles, dung, and earth.

Cross inoculations with other vegetables were not made with the forms isolated from carrots.

Symptomatology. On carrots, this pathogen produces either a crown rot or cankers on the sides of the roots (figure 7). These cankers are sometimes indistinguishable from those caused by *Rhizoctonia carotae*, and separation of the two must sometimes be made on the basis of the

microscopic characters. The presence of clamp connections in *R. carotae* serves to distinguish this latter fungus. The affected tissues under these side cankers, or in diseased crowns, are only slightly discolored, dry, and punky. Upon drying, the side cankers become hard and mumified. On the crown of the carrot the decay is more progressive and usually involves the whole root. These lesions on both the crown and the side of the root are frequently invaded by other fungi. *Gliocladium aureum*, *Penicillium oxalicum*, *P. expansum*, *Botrytis cinerea*, and *Mucor hiemalis* have been found associated with *F. roseum* on carrot roots. They soon mask the characteristic dry, punky decay of fusarium dry rot.

Reddish lesions reported by Neergaard (1937) have been observed twice on immature roots stored in home cellars in Ithaca and Erie County, New York. Isolates from these roots produced a typical, dry rot type of decay in inoculation tests. The reddish type of decay has been noted in inoculations of immature Danvers Half Long carrots when held at high humidities at 10°C.

Etiology. Wollenweber and Reinking (1935) list 75 synonyms for *Fusarium avenaceum* (Fr.) Sacc. Snyder and Hansen (1940, 1945), in recent studies on this genus, group the species of *Fusarium* into larger, more easily determinable groups. The present species would be placed under their group *Discolor*, with the name *F. roseum* Lk. ex Fr. emend. Snyder and Hansen.

Figure 7. *Fusarium* dry rot: typical lesions on side and crown of carrot root.



**Color Plates Illustrating
Six Common Storage Rots
of Carrots**

Photographs by Howard H. Lyon

Plate I



Top — Crater rot *Rhizoctonia carotae*

Bottom — Grey mold rot *Botrytis cinerea*

DISEASES OF STORED CARROTS

Plate II



Top — Cottony soft rot *Sclerotinia sclerotiorum*
 Bottom left — Black rot *Stemphylium radicinum*
 Bottom center — Crown rot *Pellicularia filamentosa*
 Bottom right — Undescribed brown side rot

Bennett (1928) has confirmed the soil-borne nature of this pathogen. It is therefore probable that infection is initiated in the field or shortly after the roots are placed in storage. Examinations made at harvest time by the writer have revealed no lesions which could be ascribed to this fungus.

In cold storages the disease seldom makes its appearance before three months have elapsed. In common storage it may appear sooner, particularly if the early storage temperatures are rather high. In the laboratory the disease usually makes its appearance in 2-3 weeks on roots held at temperatures of 60-70°F.

The disease was not observed to spread in storages, and the spores of the fungus were not isolated from the air. However, spores are invariably found on the aerial hyphae covering the lesions on the roots and are of the type that would be air-borne. The disease could easily spread from one root to another by vegetative growth.

Neergaard (1937) has shown that this fungus is carried on carrot seed produced in Europe. Isolations made by the writer from seeds grown in the United States have shown *F. roseum* to be present on seeds in this country. This constitutes an effective means of spreading this carrot pathogen to new areas.

Epiphytology. On carrot-potato-dextrose agar this organism grows over a temperature range of from 38° to 98°F, with maximum growth at 76°F. Natural infections occur over a somewhat narrower range; Lauritzen (1932) places the beginning at 44°F, and the maximum is between 60° and 70°F.

No data are available on the effect of varying humidities on infection and spread of this pathogen. Moore (1945) stated that *F. roseum* required a relatively high humidity for infection and pathogenesis in potatoes. Observations made on the appearance of this disease would indicate that humidities present in most cold and many common storages are high enough to permit decay by this organism.

Control. Maintenance of temperatures in storage at 31° to 34°F effectively controls this disease in cold storages. In common storages, during periods when temperatures cannot be kept near freezing, any practice which will keep the surfaces of the roots dry (storage in crates and hampers) and humidities low (proper ventilation) will help keep losses from this fungus to a minimum.

Brown crown rot (*Pellicularia filamentosa* [Pat.] Rogers = *Corticium solani* or *Rhizoctonia solani*)

The pathogen which causes this disease, both in its perfect and imperfect stage, is world-wide in its distribution and causes widespread losses in a number of crops (Duggar, 1915; Peltier, 1915).

P. filamentosa was first reported on New York carrots by Duggar and Stewart in 1901. These authors indicated that the rot apparently started in the tunnels of some larvae which bored into the carrot crowns. Duggar

(1915) suggested that this was probably the same disease as that reported by Kühn on carrots (not *R. crocatum* Kühn, 1858). Rolfs (1902) recorded the presence of this disease on carrots from Colorado. Heald and Wolf (1911) discovered the perfect stage on carrot stems in Texas.

During the present studies this organism has been found in the field and in both cold and common storages.

Losses from and relative importance of the disease. Aside from the damping-off phase of this disease that causes considerable damage, this pathogen produces only sporadic losses in the field and in transit when the temperatures rise above 78°F. It has never been observed to cause any appreciable damage in storage. Duggar and Stewart found only an occasional plant attacked by this pathogen in fields visited in New York State. White (1926) reported that the disease was very severe in several localities in Kansas during the 1925 season, and attributed this to the very high humidities and abnormally high temperatures present during that season. Lauritzen (1932) stated that *P. filamentosa* (*C. solani*) infects carrots at low temperatures only upon rare occasions.

White (1926) quotes Dr. Chupp (from correspondence) as saying that the disease at that time was quite prevalent in New York State and occasionally caused considerable damage.

The disease was most injurious in areas where young carrots were sold on the market with the tops still attached. Under normal growing conditions the fungus appears to be only weakly pathogenic to carrots (White, 1926) and is consequently considered to be a pathogen of minor importance.

Suscepts. Several extensive lists of the suscepts of this pathogen have been published (Rolfs, 1902; Peltier, 1915; Duggar, 1915). The very wide suscept range of this fungus includes most of the muck crop vegetables and many indigenous and escaped plants in New York State.

Symptomatology. In the field the disease is recognized by the browning and blackening on the base of the petiole and on the crown of the growing plant. As the disease progresses, a soft, brownish decay appears on the crown, forming an inverted cone-shaped lesion extending into the cylinder of the root. In the fields an infected plant would be characterized by the rotting off of the central leaves, leaving only a few of the older leaves around the edge of the decayed area.

In the storage this disease has much the same appearance; the decay in advanced stages may involve the entire crown. The affected tissues may be distinguished from botrytis grey mold rot by the darker color and the softer consistency of the rot (color plate II, bottom).

Etiology. The details of the developmental cycle of this fungus have been carefully studied on the potato; it is quite likely that the same sequence occurs on carrots. The primary source of inoculum is the soil. *P. filamentosa* is a component of the natural soil microflora, having been isolated from virgin soil (Pratt, 1918). The carrot is susceptible to attack

throughout the season. The organism may cause severe losses through damping-off of the young seedlings. A crown rot results from infections which occur later in the growing season. It is probable that all diseased roots noted in storage resulted from infection while the roots were in the field. The disease has never been observed to spread in storage, although it may logically do so by vegetative growth from one root to another.

Under certain conditions of moisture and temperature, the perfect stage of this organism is produced upon the aerial portions of the suscept. This web of mycelium which bears the basidiospores spreads over the epidermis of the petioles and leaves of the carrot with no apparent necrosis of the underlying tissues. The basidiospores produced on this mycelial mat are disseminated by the wind. Their role in the life cycle has never been established with certainty. However, it is believed that they fall on vegetable debris and initiate saprophytic cycles in the soil. The basidial stage was recorded by Heald and Wolf (1911) on carrots in Texas, and has been noted commonly in August and September on muckland carrots in New York State.

Epiphytology. White (1926) states that relatively high temperatures and very high humidities are essential for infection by this pathogen. Experiments in the laboratory have indicated that infection by *Pellicularia filamentosa* is uncertain at temperatures below 54°F, although Lauritzen (1932) stated that on rare occasions attacks could take place at 32°F. Washed roots, inoculated and held for 7 days at 70°F, showed the decay characteristic of this disease. Growth of the pathogen on agar at various temperatures shows the optimum to be near 70°F, with some growth taking place on agar at 32°F. In contrast to *Rhizoctonia carotae*, no growth was observed at 25°F after 3 weeks' incubation.

Control. Since infection by this pathogen takes place primarily in the field, control measures are first directed here. Crop rotation with non-susceptible crops, such as onions, is recommended. Better drainage or wider spacing of plants might reduce the incidence of infection in fields likely to be too moist.

Since the disease develops very slowly at 32°F, cold storage of roots will keep losses to a minimum.

Rubbery brown rot (*Phytophthora cactorum* [L. & C.] Schroet., and *Ph. megasperma* Dr.)

Seven species of *Phytophthora* have been recorded as attacking carrots: *Ph. capsici* (Wiant and Tucker, 1940); *Ph. cactorum* (Williams, et al., 1938); *Ph. drechsleri* (Tompkins, et al., 1936); *Ph. parasitica* (Godfrey, 1923); *Ph. parasitica* var. *rhei* (Godfrey, 1923); *Ph. nicotiana* (Tisdale and Kelley, 1926); *Ph. megasperma* (Dowson, 1934). Of these only *Ph. cactorum* and *Ph. megasperma* are known to attack carrots under natural conditions. The other species reported as causing decay in inoculated carrots may represent potential pathogens. However, Tucker (1933) stated: "...the

number of hosts susceptible to inoculation is of little significance. It has shown little selectivity to host wounds inoculated under favorable conditions."

During the present survey, *Ph. cactorum* and *Ph. megasperma* were found on carrots both in the field and in storage. *Ph. megasperma* caused severe losses in Middleport, Claredon, and Shelby mucklands during 1945. Dowson (1934) showed that this fungus causes serious losses under conditions of very high soil moisture. Such conditions were present over extended periods during 1945. Harvest of roots was delayed in many areas because of excessive rains. Some fields were a total loss because of the severity of this disease.

The disease occurs only sporadically in storages, and then it is usually associated with species of *Typhula*. As a storage rot, it is a disease of secondary importance.

Suscepts. Both of these species of *Phytophthora* have been found on a fairly large number of susceptibles. *Ph. cactorum* has been recorded as attacking pineapple, ginseng, rhubarb, pear, apricot, strawberry, tulip, lily, and other plants throughout the world. *Ph. megasperma* has been reported on potato, citrus, wallflower, brussels sprouts, cabbage, cauliflower, apple, tomato, beet, spinach, and other plants in Europe, England, North America, and Tasmania.

Symptomatology. In early stages of this carrot rot, the affected tissues assume only a water-soaked appearance, without discoloration of the decayed tissues. The rot is fairly firm and very wet. This symptom picture is soon changed as the tissues turn dark brown, although they remain firm and watery (figure 8). From the exterior, the rot appears very similar to the grey mold rot (see figure 2). Microscopic examinations show an abundance of intercellular mycelium composed of relatively stout, non-septate hyphae. Oospores are frequently present in the darkened layers of the cortex.

Lesions produced by *Ph. megasperma* and *Ph. cactorum* are readily invaded by *Botrytis cinerea*, *Rhizopus arrhizus*, and other more rapidly growing fungi.

Etiology. The life history of *Ph. cactorum* has recently received careful study by Blackwell (1943). Because of the similarity between these two species, it is felt that the developmental sequence of *Ph. megasperma* is essentially similar to that of *Ph. cactorum*.

Species of *Phytophthora* are considered to be inhabitants of the soil because they are most generally associated with root diseases. In the soil they are able to pass a period of dormancy as chlamydospores, oospores, or a type of resting mycelium. Spore germination is direct by the production of germ tubes. Penetration of the susceptible is believed to be purely mechanical, as demonstrated by Hawkins and Harvey (1919) for a pythium rot of potato tubers.

Sporangia are produced on the surfaces of the lesions under conditions of very high humidity. Free water and ample oxygen are apparently



Figure 8. *Phytophthora rubbery brown rot*: typical crown rot showing, water-soaked, slightly discolored decay.

necessary for the production of zoospores (Blackwell, 1943).

Secondary cycles rarely occur in storages because of the absence of enough free water essential to zoospore production. Spread from one carrot to another may possibly occur under conditions of high humidity, although it has never been observed.

Epiphytology. This disease has been observed only on carrot roots that have been subjected to flooding in the field for a period of several weeks. At the beginning of these investigations all isolates of these fungi gave negative results when inoculated into healthy carrot roots. It was found that, by soaking the roots in water for 3–4 days until they began to show signs of “drowning”, typical symptoms of the disease readily developed following inoculation. White (1945) has recently reported similar results in inoculation tests with *Ph. megasperma* on carrots.

Ph. megasperma grows over a temperature range of from 25° to 81°F, with an optimum at 60°F. At 25°F the agar medium is crystallized, and the fungus grows as a superficial mat over its surface.

Ph. cactorum does not grow at temperatures below freezing and has an optimum growth temperature of 76°F.

Control. This disease occurs primarily on growing carrot roots, its appearance in storage being dependent upon infection in the field. Control then depends on practices which will prevent roots from being subjected

to periods of excessive soil moisture in the field. Carrots grown in swampy, wet places should be harvested as soon as mature. Adequate drainage should be provided for fields that are normally wet.

Adequate ventilation during storage prevents spread of this disease (Cairns and Muskett, 1933).

Rubbery slate rot (*Pythium debaryanum* Hesse and *P. ultimum* Trow)

Middleton (1943) listed in his recent revision of this genus the following species of *Pythium* as causing carrot decay: *Pythium aphanidermatum* (Edson) Fitzpatrick, from the United States; *Pythium ultimum* Trow, from the United States; *Pythium debaryanum* Hesse, from the United States and England; *Pythium spinosum* Sawada from Formosa; *Pythium oligandrum* Drechsler from the United States; *Pythium polymastrum* Drechsler from the United States.

During the surveys made in New York, at least two species of *Pythium* were isolated from diseased carrot roots. *Pythium debaryanum* and *P. ultimum* were determined with certainty, together with several undetermined species. It is not surprising to find these pathogens attacking carrots, since they form a part of the normal soil microflora (Harvey, 1927; Meredith, 1940). The decay produced by these organisms is identical, and they are considered together in this discussion.

In storages, species of *Pythium* are rarely found and they are usually associated with other pathogens, such as *Botrytis cinera* and *Mucor* spp. In most instances, however, there was no doubt that *Pythium* spp. was the primary pathogen.

While species of *Pythium* are considered as pathogens of only very minor importance, they have been observed to attack both the seedlings and the mature roots of carrots in the field. The damping-off phase of the disease may considerably reduce the stand, particularly during cold, wet periods. *Pythium* rot of mature roots has been confined to plants subjected to periods of excessive soil moisture and low temperatures. Under these conditions this fungus causes some damage, although it is not nearly as destructive as *Phytophthora* spp.

Suscepts. *Pythium debaryanum* has been recorded on 29 susceptibles (Middleton, 1943), including a wide range of both mono- and dicotyledonous plants. *P. ultimum* has been listed as occurring on 148 susceptible species. The pythiums doubtless occur on a large number of plants in New York State.

Symptomatology. *Pythium* spp. produce a very watery type of decay in carrot roots. Pectinase secreted by the pathogen dissolves the middle lamellae. The cells of the tissues are, however, rarely completely separated, being held together by the mycelium of the pathogen, and the decay never has the mushiness of bacterial soft rot or cottony soft rot. The tissues, at first, are medium to dark brown in color and resemble grey mold rot (figure 9). They may be distinguished in later stages when the affected tissues assume a more greyish cast. Under conditions of high humidity, a white web of mycelium of the pathogen covers the affected root.

Botrytis and *Mucor* spp. commonly invade tissues affected by *Pythium* spp. and mask the characteristic symptoms of the disease.

Etiology. With the exception of the damping-off of the seedlings, the exact stage of maturity of the rot when infection takes place is not known. Diseased roots have been found in the field, and others have developed the disease during storage.

The soil is regarded as the source of primary inoculum, although there appears to be no direct evidence that the fungus vegetates in the soil. Infection may take place either while the roots are in the field or shortly after they are placed in storage.

Secondary cycles are initiated either by vegetative growth of the fungus from one root to another, or by spores produced on the fungus mycelium which sometimes covers the roots.

Epiphytology. *P. debaryanum* is fairly well adapted as a storage pathogen because of the range of temperatures over which it grows. According to Middleton (1943) this range is from 34 to 94°F, with an optimum at about 81°F. Greeves and Muskett (1936) found that pre-emergence damping-off was greatest at the lowest temperature of their experiment (43°F). Infection of carrots took place readily at 43° to 49°F in the laboratory of the writer.

Control. Because of the low minimum growth temperature of this fungus, cold storage does not prevent its development in carrots. The

Figure 9. *Pythium* rubbery slate rot: roots showing watersoaking and discoloration of affected tissue; healthy root at left.



principal means of control in storages is thus the maintenance of a humidity near 90 per cent of saturation. Storage in crates or hampers, rather than in bulk, is strongly recommended. Early harvest before heavy fall rain should be helpful.

Chemical treatment of crates is not considered essential, since the fungus is sensitive to drying and usually can be eliminated from storage containers by placing them in the sun during the period of the year when they are not in use.

Whisker rot (*Mucor spp.*)

All pathogenic species of *Mucor* and *Zygorhynchus* are included in this section because of their similarity in symptomatology and etiology. Because of this similarity there appears to be no need for separating the species found on carrots.

Mucor globosus and *M. hiemalis* have been found most commonly on carrots during the present surveys. These fungi fruit abundantly on old pieces of carrots left in the alley-ways or on the floors of the storages. The other species of *Mucor* have been found occasionally, but are not common in carrot storages.

Mucors do not cause much decay in either cold or common storages. Under conditions of artificial inoculation they readily attack carrots. It is possible that under certain storage conditions severe losses might be expected from these fungi.

Suscept. Little attention has been given in the literature to these fungi as plant pathogens. Harter (1925) lists *M. racemosus* as causing a storage decay of sweet potatoes. Panassenko (1930) has noted the following species to be pathogenic on beet in the Ukraine: *M. circinelloides*, *M. dimorphosporus*, *M. erectus*, *M. jansinii*, *M. hiemalis*, and *M. racemosus*. Most investigations on this group of fungi list the species as occurring on decaying vegetable matter in the soil (Povah, 1917).

Symptomatology. The type of decay produced by these species of *Mucor* is essentially like that caused by *Rhizopus* spp. at higher temperatures. The lesions may appear any place on the root but are commonest on the crown. The affected tissues are water-soaked and slightly brownish (figure 10b). The characteristic coarse mycelium of the pathogen soon covers the affected areas (figure 10a,c,d).

Etiology. The species of *Mucor* and *Zygorhynchus* found to attack carrots in storage include⁵: *Mucor abundans* Povah, *M. circinelloides* van Tiegh., *M. globosus* Fischer, *M. hiemalis* Wehmer, *M. jansinii* Lendner, *M. mucedo* Bainier, *M. racemosus* Fres., *M. varians* Povah, *Zygorhynchus moelleri* Vuillemin, and *Z. vuilleminii* Namyslowski.

The pathogenicity of these species has been verified by inoculation.

Little is known about the life history in the open of many of these species. It is probable, however, that their development in nature is

⁵Special acknowledgment is made to Dr. V. M. Cutter, formerly of Cornell University, for determining the species of *Mucorales* reported here.

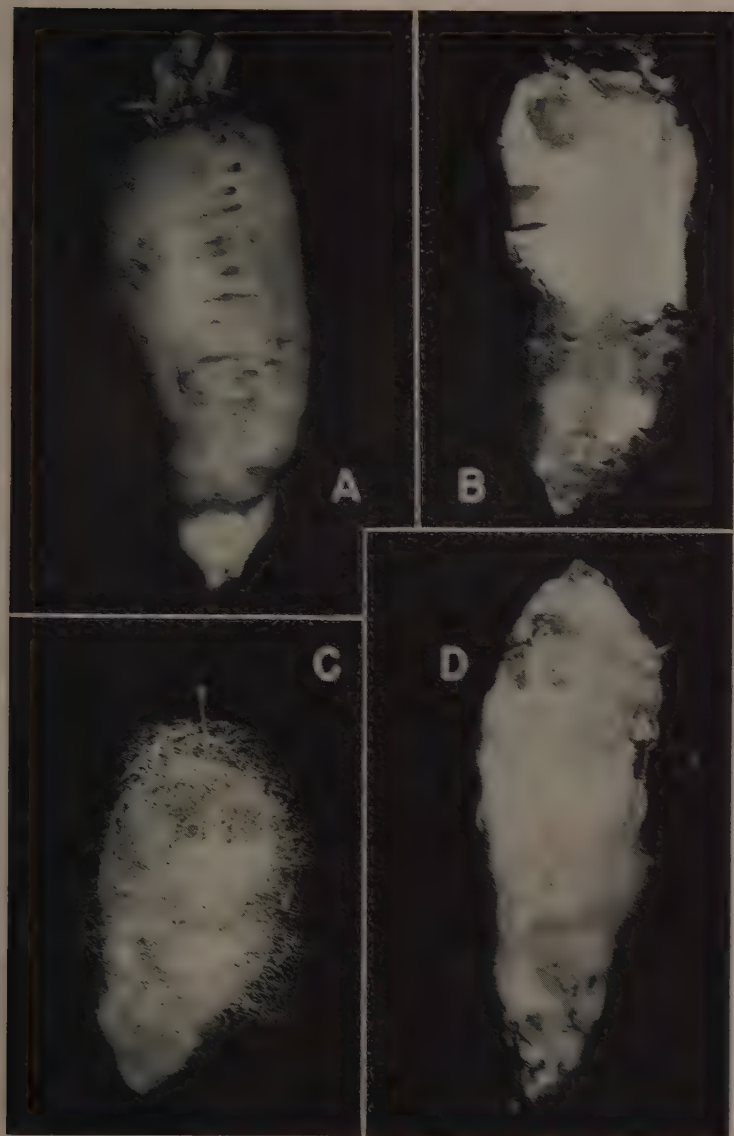


Figure 10. *Mucor* whisker rot: (a) tip rot of carrot caused by *Mucor globosus*; (b) longitudinal section of root attacked by *Mucor hiemalis*; (c) *M. hiemalis* fruiting on surface of carrot root; (d) root showing fruiting of *M. globosus*.

similar to that of *Rhizopus* sp. The fact that they have been repeatedly obtained from the soil (Gilman and Abbott, 1927) would indicate that they are normal soil inhabitants.

The pathogens are carried into storages with the particles of dirt adhering to the roots. Storage containers also serve as another source of primary inoculum, for these fungi have been commonly isolated from both empty and full containers.

These organisms develop rather slowly on carrots in storages. The lesions produced are frequently invaded by more rapidly growing pathogens, such as species of *Botrytis* and *Sclerotinia*. *Mucor* spp. may also act as "secondary invaders", preferring lesions produced by species of the Pythiaceae. Frequently, two species of *Mucor* may be found growing in the same carrot.

Mucors fruit abundantly on stored carrots and are spread readily by air-borne sporangiospores. Air analysis in storages has confirmed their presence in the air (table 2, p. 6.).

Zygospores, which are formed by most of these species, may serve to carry the pathogen over periods that are unfavorable for growth.

Epiphytology. Little is known of the factors necessary for pathogenesis and development of the diseases caused by these fungi. In culture they develop over a wide range of temperatures. Most of them grow readily at the temperatures maintained in storages. *Mucor hiemalis* and *M. globosus* grow luxuriantly at temperatures near freezing. Some isolates of *M. hiemalis* grow on agar at 25°F.

In inoculation experiments all species have produced decay at 43°F. Inoculations with *M. globosus* and *M. hiemalis*, the two species most common in carrot storages, have given positive results at 32°F.

These organisms are seldom found in the drier cold and common storages, and this probably indicates that a fairly high humidity is necessary for the development of the disease they cause.

Control. The ease with which these fungi attack carrots in the laboratory, combined with their omnipresence in carrot storages, would lead one to consider them important pathogens on carrots. This is contradicted, however, by the fact that they have never been found causing more than a fraction of a percentage loss in any storage.

The frequency with which they are isolated from storage containers makes it advisable to place these containers in a dry, exposed place during the part of the season when they are not used. Should epiphytotics of this disease occur, dipping storage containers in a suitable preservative is recommended before their use the following season.

Fungal Rots of Minor Importance

Blue-green mold rot (*Penicillium expansum* Link, *P. luteum* Sopp, and *P. oxalicum* Currie & Thom)

Penicillia rarely cause appreciable losses in carrots placed in cold storage. They are usually found on carrots that have remained in common storage for considerable periods of time and have become somewhat shriveled. They appear to prefer a low humidity and are, therefore, found more often in the common storages.

Symptomatology. A similar type of decay is produced by all these Penicillia. The affected tissues are soft and punky, only slightly darker than the surrounding healthy tissues. There are occasional white pockets of hyphae within these diseased areas, and these may be lined with the familiar green, spore-laden hyphae. The brownish, diseased tissues resulting from attacks by species of *Penicillium* have a drier and more punky consistency than the grey mold rot described earlier. Shallow lesions on the sides of the roots frequently dry until the affected tissues are very hard.

The "green mold" on the surface of the diseased tissues confirms the presence of these fungi (figure 11a). *P. expansum*, when fruiting in the dark under high humidity and low temperatures, frequently develops whitish coremia that can readily be confused with species of *Isaria* (figure 11b).

Etiology. The life histories of all of the Aspergillaceae are characterized by a series of short cycles, each initiated by the asexual spores. All three of these species have been isolated from the soil (Gilman and Abbott, 1927), and it probably constitutes the chief reservoir of inoculum. No information is available on the penetration and ingress of these pathogens into susceptible tissues. Freehand sections through diseased tissues show the pathogen to be both inter- and intracellular. Fructification begins 8 to 10 days after the first visible symptom on the root. Conidia are formed in abundance, and production of spores continues until the substrate is completely decayed or dried out.

Epiphytology. The temperature range of these Penicillia is from 32°F to 87°F; the optimum is 70°F for the species under consideration. None of these species showed growth after incubation for three weeks at 29°F. Infection takes place readily on inoculated roots placed in moist chambers and incubated at temperatures that are optimum for growth in culture. Lauritzen (1932) referred to a species of *Penicillium* that attacked carrots incubated at 107°F; none of the Penicillia found during the present survey grew at this temperature.

Observations of this disease in storages indicate that low humidities favor its development. This may be due partly to a reduced competition with more rapidly growing fungi, such as Mucorales.

Control. Control of these organisms on carrots is rarely a problem even though they are present commonly on roots, crates, and in the air. Sanitation around storages reduces the amount of inoculum and presumably the amount of decay.

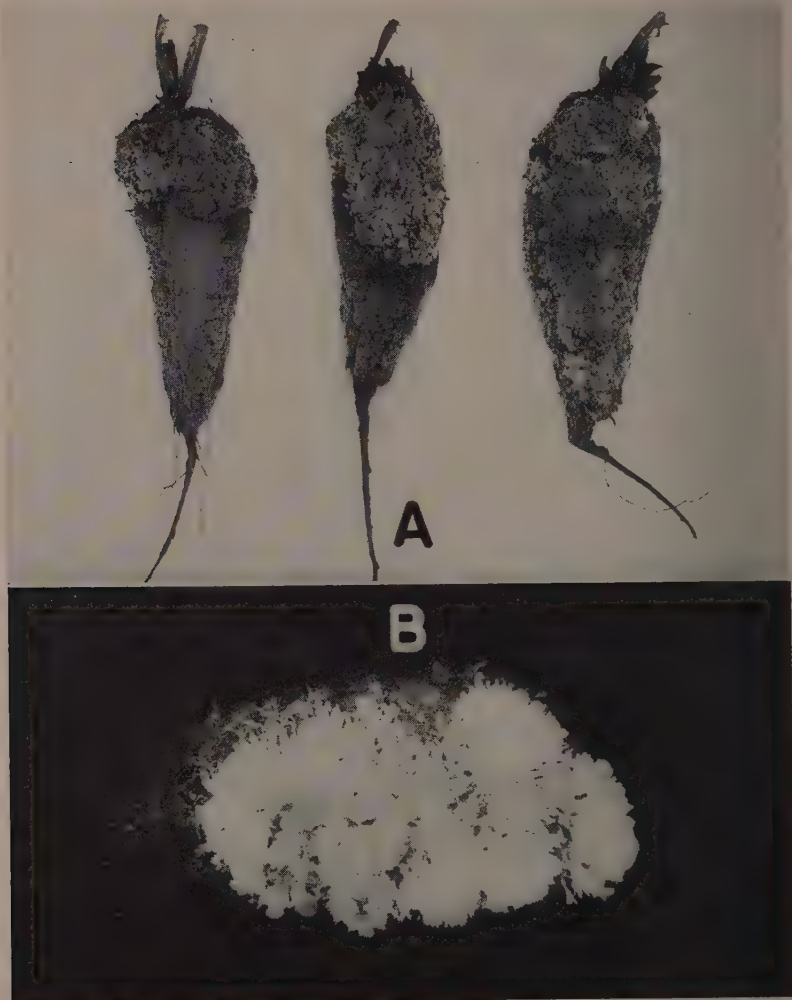


Figure 11. Blue-green mold rot: (a) typical symptoms on carrots in common storage; (b) Isaria-like coremia produced in high humidities at 32°F.

Sooty rot (*Aspergillus niger* v. Tieg.)

The sooty rot pathogen has been found commonly in every part of the world, and is particularly abundant in the tropics. It is found in warm, dry, alkaline substrate in areas where competition with the Mucorales is not very great (Rippel, 1940).

Sooty rot has been found in most common storages. The organism is strictly a wound pathogen and is prevalent on carrots with a high percentage of mechanical injury. The fungus fruits abundantly on carrots left in the fields, particularly during August and September. This disease has never been found in more than trace amounts in either cold or common storages, and is regarded as of very minor importance.

Suscepts. *Aspergillus niger* has been reported on an unbelievably wide range of substrates. Records show its presence in the human ear and lung, in spoiling raw sugar, and in rancid butter and other fats. Floating and submerged mycelia of this fungus have been found in many chemical solutions. It is also found in grains, forage products, spoiled fruits and vegetables, cotton fabrics, leather, and decaying vegetation in the field. It is isolated commonly from the soil (Thom and Raper, 1945). It has been isolated by the writer from rotting apples and pears in New York storages. Shapavolov (1919) records this fungus as a pathogen on potatoes. Isolates from pear, apple, soil, and carrot all proved to be pathogenic on carrot.

Symptomatology. Typical lesions caused by this fungus may be found on any part of the root. They are shallow (usually only a few millimeters deep), tough, slightly discolored, and the surface of the affected area is covered by the black fructifications of the pathogen. At high humidities the decay may penetrate 2 or 3 centimeters into the carrot (figure 12a, b).

Lesions produced by this organism are rapidly invaded by species of *Mucor*, *Zygorhynchus*, and *Botrytis* that completely mask the original aspect of the affected area.

Etiology. *Aspergillus niger* van Tieghem was first described in 1877. The life history of this pathogen can be pieced together from the records of its occurrence throughout the season. The almost universal presence of the organism on carrot roots during growth or storage would seem to show that the primary cycles may be initiated whenever conditions favor pathogenesis. Within a few days to a week after its establishment, the fruiting structures appear and initiate a series of secondary cycles.

A. niger remains alive in the soil during the winter. Present studies indicate that the fungus will remain viable in culture for at least two months at 3°F. It may also remain viable in the storage house from one season to another.

Epiphytology. Temperature is probably the most important circumstance governing the pathogenesis of this fungus. It grows over a fairly wide range of temperatures; the optimum lies at 92°F. Infection studies by the writer show that this temperature is very close to the optimum for penetration and pathogenesis by this organism. During these tests,

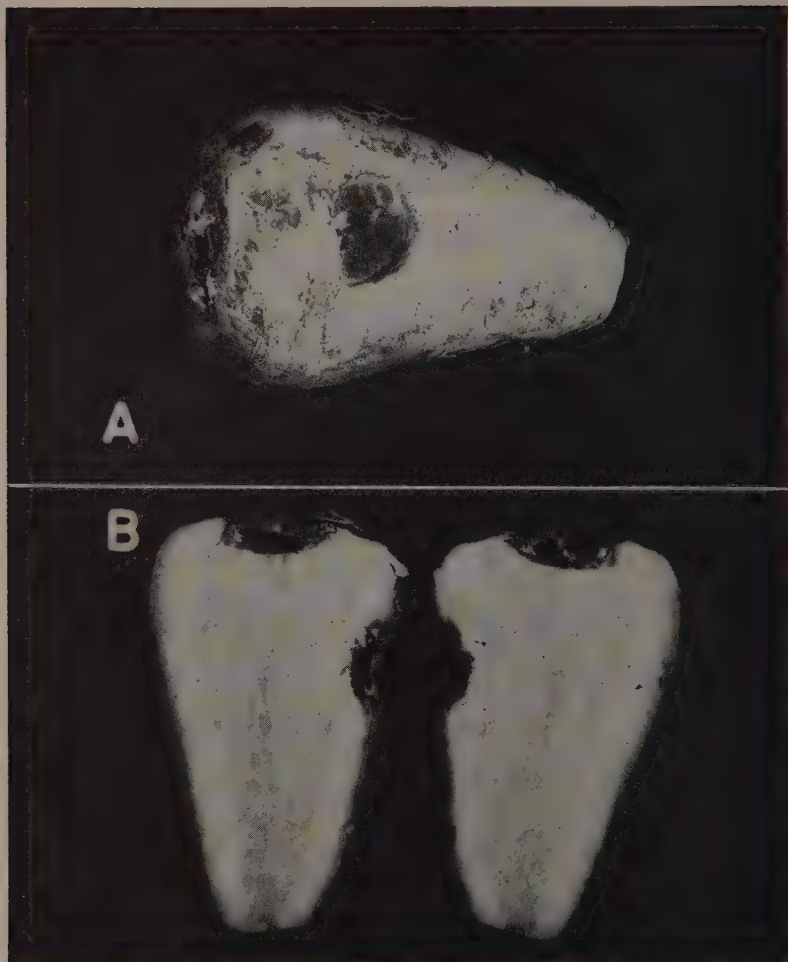


Figure 12. Sooty rot: (a) typical lesion on side of carrot root; (b) longitudinal section through carrot root, showing shallow, sunken canker.

inoculated carrots held at a temperature of 32°F never have become infected.

At the optimum temperature of 92°F for *A. niger*, high humidity so encourages the growth of Mucorales as to prevent the growth of the former pathogen. The presence of sooty rot on carrots left lying in the sun in the fields is presumably due to the favorable combination of high temperature and low humidity.

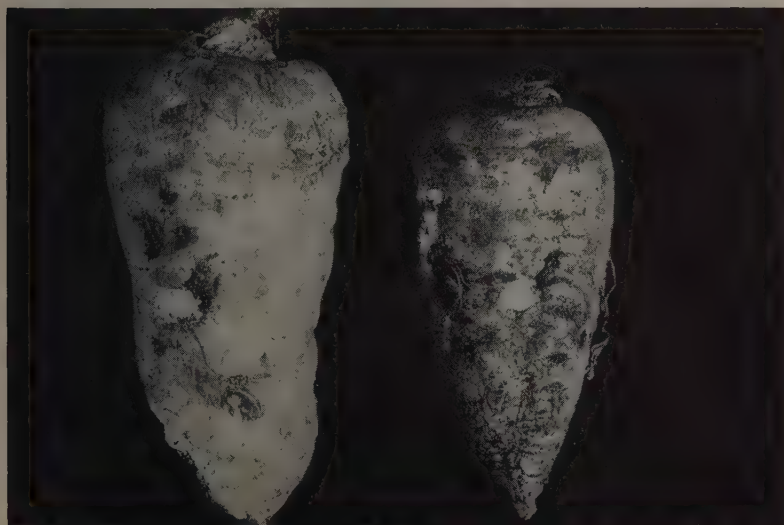
Control. This organism is readily controlled by care in handling the roots to prevent excessive injury and bruising. Storage at temperatures near freezing prevents infection and development of this pathogen.

Hard rot (*Gliocladium aureum* Rader)

Hard rot has been found causing losses in scattered lots of stored carrots in the cold storages of several counties. This disease has been confused with other diseases. *Gliocladium* hard rot actually causes only a very small amount of damage and under standard storage conditions should never cause severe losses (Rader, 1948).

Symptomatology. The symptoms produced on carrots by this organism are very similar to those produced by *Fusarium roseum*, with which it is frequently associated. The lesions on carrots appear as shallow, slightly sunken cankers on the side of the root (figure 13). The decay is generally hard and leathery, rarely extending into the tissues more than a few millimeters, and the affected tissues are light brown. Dried out cankers

Figure 13. *Gliocladium* hard rot: shallow, hard lesions on roots taken from cold storage.



caused by *F. roseum* are practically indistinguishable from those caused by this pathogen. In roots inoculated by the usual "well" method and incubated in moist chambers at 41°F, the decay progresses slowly, turning the tissues brown and leaving the diseased area somewhat softer than the unaffected portion. The affected area is frequently covered with a thick mat of mycelium. The lesions are readily invaded by *Botrytis cinerea*, *Mucor hiemalis*, or *Fusarium roseum*, which quickly mask the characteristic symptoms of this disease.

Etiology. No data are available on the exact life history of this fungus. Its similarity to species of *Penicillium*, both in distribution and in cultural requirements, would seem to indicate a similar seasonal development; that is, a succession of cycles on roots throughout the year, whenever the suspect is present and proper environmental conditions exist.

Epiphytology. Studies on growth of this fungus in pure culture indicate that the optimum temperature lies between 70° and 76°F, the minimum below 27°F, and the maximum between 92° and 98°F. Infection has been obtained regularly on inoculated roots incubated in moist chambers at 43° and 70°F.

Control. Gliocladium hard rot is readily controlled by storage of roots at 32°F. Even at higher temperatures little damage has been observed which could be directly attributed to this fungus.

Buckshot rot (*Typhula intermedia* Appel and Laub., *T. umbrina* Remsberg, and *T. variabilis* Riess.)

Typhula spp. are regarded by most workers as weak pathogens on vegetables (MacDonald, 1934; Rambousek, 1924). Epiphytotics are rare and are restricted in area (Schmidt, 1933).

During the present study, *Typhula* was found frequently on carrots in cold storage. Estimated losses ranged from a trace in most cold storages to a fraction of 1 per cent during the 1944-45 storage season. The organisms were found less frequently in the 1945-46 season. These surveys, together with subsequent studies, indicate that *Typhula* spp. are of very little importance as decay organisms in stored carrots.

Suspects. The three species of *Typhula* here reported on carrots have been found on a variety of substrates. *Typhula intermedia*, described in this bulletin for the first time as a pathogen, usually has been collected upon fallen overwintering leaves, petioles, and herbaceous stems (Remsberg, 1940). *T. umbrina* has been known previously from only two collections on Iris rhizomes and stored turnips made at Ottawa, Canada (Remsberg, 1940). *T. variabilis* is more commonly known on vegetables, and has been reported on asparagus, beets, potatoes (Schmidt, 1933), and celery (Remsberg, 1940). The writer has also found *T. variabilis* on carrots and on celery in a Fairport storage. *Typhula gyrans* Batsch ex. Fr. has been described by Jørstad (1928) on carrots, cabbage, and celery from Norway; it was not found during the present surveys.

Symptomatology. Because the lesions produced by *Typhula* spp. are readily invaded by other fungi, and *Typhula* spp. may also act as secondary organisms, carrots are rarely found in storages with lesions typical of these organisms. *T. intermedia* and *T. variabilis* commonly cause a shallow, slightly brownish lesion with the edges not markedly differentiated from the healthy tissues (figure 14a, c). The decay associated with *T. umbrina* is darker and of a more stringy consistency (figure 14b, c). In the laboratory all three species produce a very shallow, cheesy decay.

Sclerotia are usually found associated with the decay in storage, and serve to confirm the presence of these organisms. The sclerotia appear as small, spherical, brown to black bodies, 0.5 to 6 mm in diameter, superficial or sunken in the lesions. They are distinguished from the sclerotia of *Sclerotium rolsii* by the absence of a white aerial mycelium on the surface of the carrot (figure 14a, b).

Etiology. Very little actual experimental data are available on the life history of these organisms that attack vegetables. MacDonald (1934), working on a related species (*T. gyrans*), has made some careful observations on its seasonal development. Resistant sclerotia, formed during the cool temperatures of spring and fall, are capable of withstanding the heat and drouth of the summer and the cold of the winter. During periods favorable for growth, these sclerotia may germinate to produce sporophores or a new thallus. They can survive for a considerable length of time in soils containing a reasonable amount of organic matter (MacDonald, 1934).

The pathogen is carried into the storages in the fall, either in the soil particles adhering to the roots or as incipient infections. Observations made at harvest time have revealed no lesions that could be ascribed to these fungi. *Typhula variabilis* has been isolated from the containers used to store carrots, thus indicating a second source of primary inoculum. The disease makes its appearance on carrots after 2 to 3 months of storage. The buckshot rot has never been observed to spread in storage.

Sporophores are produced from sclerotia on the soil in areas exposed to sunlight (Remsberg, 1940). Basidiospores, thus produced, germinate readily on decaying plant material and produce a new thallus. MacDonald (1934) noted that *T. gyrans* was heterothallic. Other species have not as yet been investigated.

From the available data it appears that species of *Typhula* existing in the soil develop in cool weather during the fall and are carried into the storages at harvest time as contaminants on the roots. Infection presumably takes place after the carrots are placed in storage. Occasional infections may take place from inoculum carried with the crates. The decay progresses very slowly, and is first noted as small, depressed lesions containing a few small, round, brownish sclerotia.

Epiphytology. Low temperatures and relatively high humidities are essential for the development of *Typhula* (Remsberg, 1940; MacDonald,

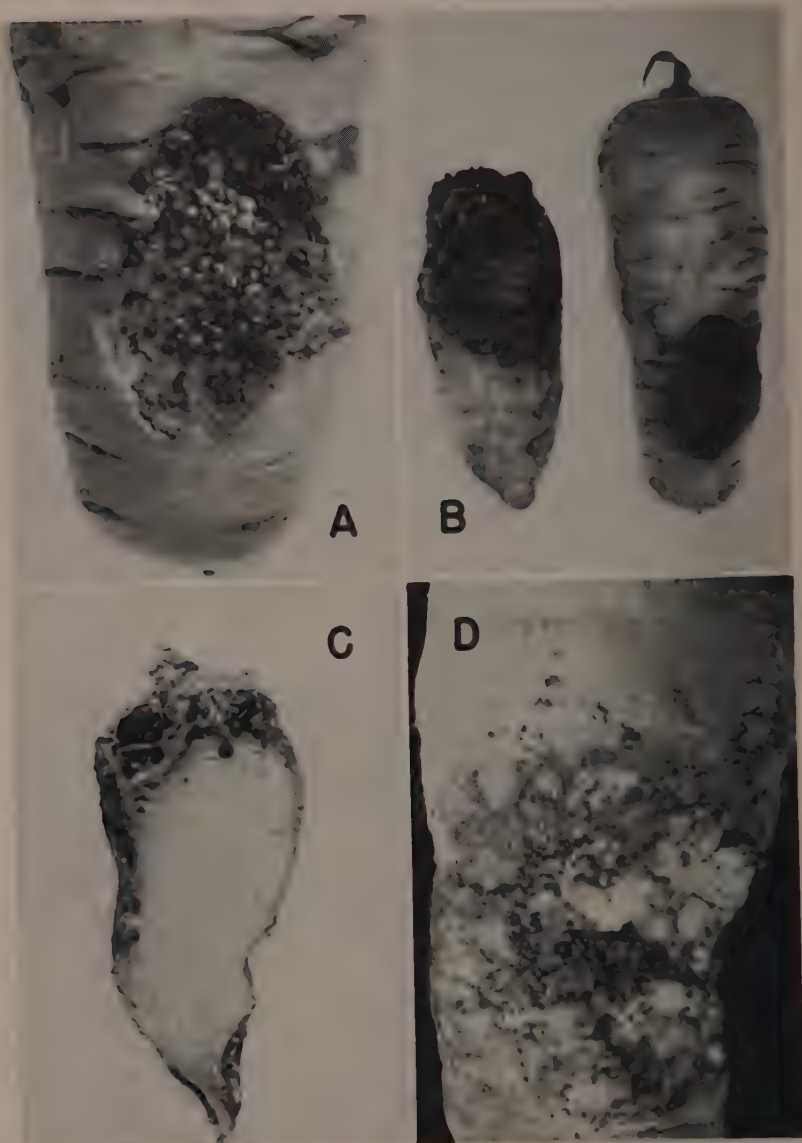


Figure 14. *Typhula* buckshot rot: (a) typical lesions caused by *Typhula intermedia*; (b) soft rot caused by *T. umbrina*; (c) longitudinal section through diseased roots, showing sclerotia of *T. umbrina* within affected tissue; (d) lesion caused by *T. variabilis*.

1934). Remsberg listed the temperature range of these fungi as 32°–70°F, with an optimum between 54° and 65°F.

The low minimum temperature for these organisms makes them particularly suited to growth in cold storages, but their weak pathogenicity keeps them from reaching epiphytotic proportions there.

The sclerotia produced by these fungi are particularly resistant to temperature and moisture changes, and permit survival of the fungus during the summer months.

Little is known about the effect of lowered humidity upon the fungus thallus. MacDonald (1934) indicated that 5 minutes' exposure to laboratory air is enough to dry up the sporophores completely. It is probable that the vegetative hyphae are equally sensitive.

Control. The minor importance of this disease makes any special control measures seldom necessary.

Discussion

A network of cold and common storages across the country provides our vegetable processors with a supply of carrots and other vegetables throughout the winter canning season. In spite of modern refrigeration, there is a considerable loss of edible products before the end of each storage season. These losses are frequently multiplied when produce that has developed a small amount of disease in storage is being shipped. The consumer obviously must underwrite this wastage, a cost ultimately reflected in what he pays for the finished products.

This bulletin aims primarily to describe the various storage rots encountered and to suggest only briefly a few things that may aid in reducing losses. For those interested in the identification of the 16 diseases described, a key (page 63) has been provided that may be helpful. Unfortunately, very little experimental work has been done to compare different procedures that might extend the storage period of carrots safely.

The responsibility for holding losses to a minimum would seem to rest jointly upon the shoulders of the grower and the storage operator. As this bulletin emphasizes in the section on sclerotinia cottony soft rot, the important diseases with their origin in the field point to the necessity of a wise rotation in which carrots do not follow carrots or any other crop subject to similar diseases. When carrots follow either carrots, beans, lettuce, celery, or cabbage, there is added likelihood of trouble from cottony soft rot, grey mold rot, bacterial soft rot, or of licorice rot when carrots follow celery. If carrots follow cereals, potatoes, spinach, onions, corn, pasture or hay crops, the danger from these major storage diseases should be much reduced.

Good drainage of the land is probably important in reducing the incidence of most diseases, particularly those whose causal organisms thrive best on a water-soaked carrot, such as species of *Pythium*, *Phytophthora*, *Botrytis*, and soft rot bacteria. It may be wise to pull carrots early if previous experience and poor drainage indicate that roots may be in prolonged contact with wet soil.

Harvested carrots should not be left out in the rain and should be stored in crates rather than bulk. Crates or baskets should either be new or should be disinfested by steam, sunshine, or by dipping in a suitable fungicide. Out of 625 cultures made from as many crates and baskets in three cold storage rooms, a pathogenic species of *Mucor* was obtained 53 times, of *Botrytis* 43 times, of *Penicillium* 18, *Rhizoctonia carotae* 17, *Sclerotinia sclerotiorum* 16, *Pythium* species 5, a *Fusarium* 3, an *Alternaria* and a *Typhula* each once. Storage of a fresh clean crop in old containers without disinfesting or decontaminating them first would therefore seem to be inviting trouble. Even a few days in the hot sun helps to eliminate many organisms.

Growers could use more care not to fill crates in the field so full that their contents get crushed, broken, or unduly bruised during the several loadings and unloadings that must take place before they are finally stacked in the storage rooms. There is evidence that soil adhering to crate bottoms may cause the top layer of carrots in the crate below to develop more crater rot than the others. Baskets are not thought to be as good as crates for stacking with a minimum of crushing.

That bruised carrots are subject to inoculation with the spores of many pathogenic fungi after they are placed in cold storage was shown in a series of analyses, by appropriate methods, of 18 three-cubic-foot samples of air from three rooms in one storage near Rochester. Viable spores of species of *Aspergillus*, *Botrytis*, *Gliocladium*, *Mucor*, and *Penicillium* were clearly present. Soil laden with many pathogenic microorganisms also may come in contact with these injured areas and inoculate the roots with various pathogens.

The writer has attempted control of the serious storage diseases by fungicidal dips before storage, but out of 40 chemicals tried against 21 carrot pathogens, none proved worth recommendation.

Recent work in Finland by Mukula (1950) indicates that one or two fungicidal powders, dusted over bushel crates of carrots before they were placed in common storage, helped to reduce loss from grey mold rot. These might not find favor with American processors, who must have a product free from chemicals imparting objectionable odors, flavors, or off-colors to the final product. Investigations of the possible chemical control of the major storage diseases should be pursued further.

It has been repeatedly pointed out that many pathogens are able to make some growth even below the freezing point (*Rhizoctonia carotae*, *Sclerotinia sclerotiorum*, *Botrytis cinerea*, and *Gliocladium aureum*). Each degree above the minimum enables them to grow faster. For maximum storage in the best possible condition carrots should therefore be cooled as soon as possible after harvest to the lowest point at which they will remain unfrozen; this is believed to be between 30 and 31°F. It is unfortunate that a frozen carrot does not process satisfactorily; the problem of keeping them for long periods would otherwise be simpler. In this con-

nection it is noteworthy that carrots stacked in rooms held close to freezing may not come down to the temperature of the room for several weeks, or even months. They generate considerable heat by their own respiratory processes, heat that is a definite part of the normal refrigeration load. It is the chief reason why the carrots lag several degrees in coming down to the desired room temperature. In the absence of forced air circulation, the higher the room and the stack of crates, the greater the difference in carrot temperature between top and bottom crates; there is often a difference of 2 to 4 degrees F. The carrots may be 3° to 5°F above room temperature at the top of the stack. This may be why carrots seem to keep better in low-ceilinged rooms than in high ones. For the first week or two of storage, rooms could perhaps be held at lower temperatures than they commonly are, with an eye on the internal temperature of the carrots as well as on the air of the room. Precooling in ice water is being tried, but general opinion favors storing most produce, including carrots, unwashed for the long pull. Forced air circulation, as employed in the latest apple storages, would probably help cool carrots more rapidly and permit a temperature between 30° and 31°F to be safely and evenly maintained throughout the rooms.

The control of humidity is important because it is closely related to water of condensation or surface film in which spores can germinate. It would seem that a low humidity, between 80 and 90 per cent, would be desirable during the early part of the storage period when the temperatures are not yet down to 31°F. Storage in crates is preferable to bulk storage in both cold and common store rooms, because freer air circulation through a stack of crates helps reduce both surface moisture and temperature.

Summary

Carrots decay in both cold and common storages. The decay which appears in either type of storage is dependent upon (1) the land upon which the crop was grown (drainage, previous crops); (2) the care used in handling the crop on its way to storage; and (3) the conditions under which the crop is stored (temperature, humidity, piled in crates or bulk, and so forth). The diseases described in the present bulletin are those commonly found in cold and common storages in New York state. They have been grouped as to their observed importance.

Rots of major importance

Bacterial soft rot caused by *Erwinia carotovora* was found in the field and was more or less prevalent in common and pit storages where temperatures were not low enough to hold it in check. This disease is not important while carrots are in cold storage. Rotting is more rapid in the core, and is characterized by its watery, shiny consistency and lack of visible fungus growth. A small incidence of this disease in any type of storage must be regarded as serious, since a rise in temperatures during subsequent handling or shipping will produce conditions optimum for its destructive spread.

After an abundance of moisture before harvest, grey mold rot (*Botrytis cinerea*) can become serious both in the field and later in either common or cold storages. The presence of the fluffy, grey-brown mold and/or black, irregularly shaped sclerotia on the rotting surface of the roots are each good diagnostic characters of this disease. As the season progresses, even in cold storages, losses increase, for the fungus can grow and propagate at subfreezing temperatures. Six species of *Botrytis* were shown to be capable of rotting carrots. Many viable spores of this fungus have been found in the air of cold-storage rooms.

Cottony soft rot of carrots can be caused by 8 different species of the fungus *Sclerotinia*, although only one was found in New York (*S. sclerotiorum*). The dense, heavy, white mycelium and large, creamy to black, round sclerotia on the surface of the rotting tissue serve to distinguish it from the other rots. It can spread through containers and grows rather well even at freezing temperatures. In common storages where temperatures are permitted to rise and in transit this disease is responsible for serious losses. Proper crop rotation or use of cyanamid in the field, coupled with prompt cooling of the roots to 31°F as soon as they are placed in storage should keep this disease to a minimum.

In some seasons crater rot caused by *Rhizoctonia carotae* is by far the most serious cold-storage carrot disease, both because of its widespread distribution and its ability to grow at subfreezing temperatures. It is distinguished by the shallow, cream-white, sunken pits or lesions, anywhere on the root, that may give rise later to a heavy growth of dense white mycelium, very much like *S. sclerotiorum* but without the large sclerotia. This disease is known principally in cold storages; it is rare in the common storages. This suggests that its development is dependent upon the higher humidity in the cold storages. Prompt chilling and holding of the carrots down to 31°F and piling of crates or baskets to permit the free circulation of air to keep the humidity down, is all that can be suggested for control at present.

Licorice rot, caused by the fungus *Centrospora acerina*, is a relatively new disease on carrots and is probably confined at present to muckland crops grown in rotation with celery. On celery this fungus produces a black crown rot often serious in northern cold storages. The fungus lives over winter in the muck soil. On carrots it causes dark brown to black, more or less diamond-shaped lesions anywhere on the roots. Affected areas have water-soaked margins and no visible surface mycelium. *Centrospora* sp. can develop at low temperatures, but so far licorice rot is of less general importance than the first 4 diseases just described.

Black rot of carrots (*Stemphylium radicinum*) can develop anywhere on the root. Frequently infection takes place in the crown where the ensuing decay penetrates rather deeply. On the sides of the roots the lesions are circular, shallow, almost jet black, and slightly depressed; they are distinguished from the licorice rot by the black discrete margins of the

decayed tissues. In high humidities the lesions may be covered with a dark, olivaceous, fluffy fungus growth. While it can be found in cold storages at temperatures down to 31°F, this disease is much more prevalent in common storages. Since the spores of this fungus need water on the surface of the carrot root to germinate, it would seem probable that incipient infections occur on the root prior to storage; this emphasizes again the necessity of proper crop rotation. The fungus is seed-borne and can cause seedling blight and leaf spotting in the field similar to the well known carrot blight, so a spray program with nabam or zineb should be helpful in its control on the foliage.

Rots of secondary importance

The woolly soft rot caused by three species of *Rhizopus* is not a problem in cold storages, and causes losses in common storages only when temperatures are permitted to rise abnormally high. However, under conditions frequently prevalent during transit, these fungi can reduce a carload of carrots to a complete loss. The abundance of coarse woolly white mycelium over the brownish water-soaked lesions anywhere on the root generally distinguishes this rot.

Dry rot due to *Fusarium roseum* commonly occurs on the crown, where it causes extensive decay, or on the side of the root, where it forms a dry, sunken, punky rot, slightly discolored to reddish in color. At one stage it may have white mycelium growing on the lesions and be almost indistinguishable from crater rot. But the fungus, which is seed-borne, is unable to grow below 35°F, so it is not important in cold storages.

Brown crown rot caused by *Pellicularia filamentosa* (*Rhizoctonia solani*) may start in the field as a soft brownish decay of the bases of the central leaves, and in storage this brown rot progresses down into the central core of the root. While the decay is similar to the grey mold rot it is distinguished by its darker color and softer consistency. Temperatures above 45°F are necessary for infection and rapid development of this disease, so it is checked by cold-storage temperatures.

Roots that have been subjected to flooding for a few weeks in the field before harvest may develop rubbery brown rot caused by soil-inhabiting species of *Phytophthora* (*P. megasperma* or *P. cactorum*). The causal fungi can grow at temperatures as low as 25°F. The decayed tissues are at first water-soaked, then become brown; the rot usually begins at the crown of the root.

Another similar rot is caused by the common soil-inhabiting fungus *Pythium debaryanum*. Its slate grey color is often overrun by *Mucor* spp. in common storage and by *Botrytis* spp. in cold storage, so its identity is soon obscured. Infection by the forgoing species of *Pythium* and *Phytophthora* depends upon subjection of the roots to extremely wet conditions in the field; hence control is primarily a matter of preventing such conditions before harvest.

Rots of minor importance

This group includes several rots caused by weak pathogens or fungi that are unable to grow at storage temperatures.

The blue-green mold, *Penicillium expansum*, is found everywhere on decaying plant and animal debris. It produces an abundance of air-borne spores that make it nearly ubiquitous in its distribution. It is the cause of one of the most common secondary rots which follows those listed here as of major importance, particularly when humidities have gone down late in the storage season.

Sooty rot, caused by *Aspergillus niger*, is strictly a high-temperature decay and is often seen on culls left in the field in August and September. The fungus survives the winters on trash in the fields and is distinguished by its olive black color and lack of conspicuous fungal growth.

Hard rot caused by *Gliocladium aureum* is difficult to distinguish from dry rot caused by *Fusarium* sp., being shallow, hard, and leathery. The pathogen can grow at temperatures as low as 27°F but is apparently weakly pathogenic on carrots, and therefore rarely troublesome.

Buckshot rot gets its name from the small, brown, round sclerotia formed by the causal fungi, *Typhula gyrans*, *T. intermedia*, or *T. variabilis*. These bodies are usually formed on the surface of the shallow, light-brown lesions, which cover the cheesy decay that characterizes the rot. It is more of a curiosity than a menace to carrots in cold storage, where it occurs chiefly on the stubs of leaf stalks.

Among the many mold fungi that grow on crates and baskets were a number of pathogens, such as species of *Botrytis*, *Sclerotinia*, *Rhizoctonia*, *Penicillium*, *Pythium*, *Fusarium*, *Mucor*, and *Rhizopus*. This emphasizes the wisdom of disinfecting all containers each year before using them to store carrots.

Many infections are believed to occur in the field before or during harvest, but there are further opportunities for them after carrots are put in cold storages. An analysis of 18 samples of air revealed the presence of spores of species of *Botrytis*, *Gliocladium*, *Mucor*, *Penicillium*, and *Aspergillus* in the rooms. The most serious of these fungi in both common and cold storages is *Botrytis*.

Many rots of carrots found in storages are difficult or impossible to diagnose because so many different fungi and bacteria grow on them simultaneously. Growers, storages operators, and processors can usually determine the principal cause of decay in stored carrots by the symptoms on newly attacked roots.

References

- Beach, W. S.
1922. Diseases of certain truck crops caused by *Sclerotinia* and *Botrytis*. In Pa. Agr. Exp. Sta. Bul. 170:18.
- Bennett, F. T.
1928. On two species of *Fusarium*. *F. culmorum* (W.G. Sm.) Sacc. and *F. avenaceum* (Fries) Sacc. as parasites of cereals. Ann. Appl. Biol. 15:213-244.
- Blackman, V. H.
1925. Physiological aspects of parasitism. Brit. Assoc. Adv. Sci. Rept. 92:233-246.
- Blackwell, E.
1943. The life history of *Phytophthora cactorum* (Leb. and Cohn) Schroet. Brit. Mycol. Soc. Trans. 26:71-89.
- Blakeslee, A. F.
1904. Studies in the Mucorineae. I. Sexual reproduction. Amer. Acad. Arts and Sci. Proc. 40:205-319.
- Bolle, P. C.
1924. Die durch Schwärzepilze (Phaeodictyae) erzeugten Pflanzenkrankheiten. Meded. Phytopath. (Holland) 7:1-77.
- Boyle, C.
1921. Infection by *S. libertiana*. Ann. Bot. 35:337-347.
- Brown, W., and Harvey, C. C.
1927. Studies in the physiology of parasitism. X. On the entrance of parasitic fungi into the host plant. Ann. Bot. 41:643-662.
- Burkholder, W. H., and Smith, Wilson L. Jr.
1949. *Erwinia atroseptica* (Van Hall), Jennson and *Erwinia carotovora* (Jones) Holland. Phytopath. 39:887-897.
- Cairns, H., and Muskett, A. E.
1933. *Phytophthora megasperma* causing pink rot of the potato. Nature 131:227.
- Christoff, A., and Christova, E.
1939. Some plant diseases new to Bulgaria (4th contribution). [In Russian.] Soc. Bot. Bulgarie. Bul. 7:39-40.
- Chupp, Chas.
1925. Manual of vegetable diseases. 647 pages.
- Ciferri, R.
1927. Notae mycologicae et phytopathologicae. Serie II. n. 1-15. Riv. Patol. Veg. 17:209-294.
- Coemans, M. E.
1860. Recherches sur la genest et les metamorphoses de la *Peziza sclerotiorum* Lib. Acad. Roy. Sci. Belg. Bul. 119-61.
- Cunningham, G. H.
1927. Fungus diseases attacking artichokes. New Zeal. J. Agr. 34:402-408.

De Bary, A.

1886. Über einige Sclerotien und Sclerotienkrankheiten. Bot. Zeit. 44:377-387, 393-404, 409-426, 433-441, 449-461, 465-474.

Dickson, Frank

1930. Studies on *Sclerotinia sclerotiorum* (Lib.) de Bary. Thesis for the degree of Ph.D., Cornell University. (Unpublished.)

Dowson, W. J.

1934. *Phytophthora megasperma* Drechsler in Tasmania. Brit. Mycol. Soc. Trans. 19:89-90.

Duggar, B. M.

1915. *Rhizoctonia crocorum* (Pers.) DC. and *R. solani*. (*Corticium vagum* B. & C.) with notes on other species. Mo. Bot. Garden. Ann. 2:403-458.

Duggar, B. M., and Stewart, F. C.

1901. The sterile fungus *Rhizoctonia* as a cause of plant diseases in New York. Cornell Univ. Agr. Exp. Sta. Bul. 186:51-76.

Gilman, J. C., and Abbott, E. V.

1927. A summary of the soil fungi. Iowa State Coll. J. Sci. 1:225-345.

Godfrey, G. H.

1923. A phytophthora foot rot of rhubarb. J. Agr. Res. 23:1-26.

Greeves, T. N., and Muskett, A. E.

1936. A temperature study of *Pythium* attack on swede seedlings. Ann. Appl. Biol. 23:264-270.

Groves, J. W., and Skolko, A. J.

1944. Notes on seed-borne fungi. II. *Alternaria*. Canad. J. Res. C. 22:217-234.

Gutner, L. S., and Khokhrjakov

1940. A study of cultivated plant diseases on the Kolsky Peninsula. [In Russian.] Internatl. Bul. Plant Protect. 1-2:245-250.

Harter, L. I.

1925. A physiological study of *Mucor racemosus* and *Diplodia tubericola*, two sweet potato storage rot fungi. J. Agr. Res. 30:961-969.

Hartig, Robert

1880. Der Ahornkeimlingspilz, *Centrospora acerina*. Untersuch. Forst-Bot. Inst. München. 1:58-61.

Harvey, J. V.

1927. A survey of water molds occurring in the soils of Wisconsin, as studied during the summer of 1926. Wis. Acad. Sci. Trans. 23:551-562.

Hawkins, L. A., and Harvey, R. B.

1919. Physiological study of the parasitism of *Pythium debaryanum* Hesse on the potato. J. Agr. Res. 18:275-298.

Heald, F. D., and Wolf, F. A.

1911. A plant disease survey in the vicinity of San Antonio, Texas. U. S. Dept. Agr., Bur. Plant Indus. Bul. 226:1-129.

- Hirta, E.
1927. Studies on the rotting disease of *Amorphophallus rivieri*. [In Japanese.] Mitt. Landw. Versuchsstat. Tokyo. 48:1-45. (Abstr. in Rev. Appl. Mycol. 7:296.)
- Jensen, C. W.
1912. Fungous flora of the soil. Cornell Univ. Agr. Exp. Sta. Bul. 315:414-501.
- Johnson, D. E.
1930. The relation of the cabbage maggot and other insects to the spread and development of soft rot of the Cruciferae. Phytopath. 20:857-872.
- Jones, L. R.
1901. *Bacillus carotovorus*, the cause of the white rot of carrots. Centbl. Bakt. u. Par., 2 Abt. 7:12-21, 61-68.
- Jørstad, I.
1928. Report on plant diseases in agriculture and horticulture. V. Economic horticultural plants. [In Danish.] 68 pages. Oslo.
- Kühn, J.
1858. Krankheiten der Kulturgewächse. 312 pages. Berlin.
- Lauritzen, J. I.
1926. The relation of black rot to the storage of vegetables. J. Agr. Res. 33:1025-1041.
- Lauritzen, J. I.
1932. Development of certain storage and transit diseases of carrots. J. Agr. Res. 44:861-912.
- Link, G. K., and Gardner, M. W.
1919. Market pathology and market diseases. Phytopath. 9:497-520.
- MacDonald, J. A.
1934. The life history and cultural characters of *Typhula gyrans* (Batch) Fries. Ann. Appl. Biol. 21:590-613.
- McLean, J. G., and Walker, J. C.
1938. New wilt disease (*F. avenaceum*) of potato. In Wis. Agr. Exp. Sta. Bul. 440:40-57.
- Machacek, J. E.
1928. Studies in the association of certain phytopathogens. Quebec Soc. Protect. Plants. Ann. Rept. 20 (1927-1928):16-63.
- Malençon, G., and Delécluse, R.
1937. Champignons pathogenes observés au Maroc. Soc. Sci. Nat. Maroc. Bul. 17:132-144.
- Meier, F., Drechsler, C., and Eddy, E.
1922. Black rot of carrots caused by *Alternaria radicina* n. sp. Phytopath. 12:157-166.
- Meredith, C. H.
1940. A quick method of isolating certain Phycomycetous fungi from the soil. Phytopath. 30:1055-1056.

Middleton, J. T.

1943. The taxonomy, host range and geographic distribution of the genus *Pythium*. Torrey Bot. Club. Memoir. 20:1-171.

Moore, F. J.

1945. A comparison of *Fusarium avenaceum* and *F. caeruleum* as causes of wastage in stored potato tubers. Ann. Appl. Biol. 32: 304-309.

Mounce, I., and Bosher, J. E.

1943. Seedling blight of carrot caused by *A. radicina*. Sci. Agr. 23: 421-423.

Mukula, Jaakko

1950. On chemical prevention of storage rot in carrots. [In Finnish; English summary.] Sci. Agr. Soc. Finland. J. 22:86-92.

Neergaard, P.

1937. Attacks of *Alternaria radicina* on celery and carrot. Roy. Vet. and Agr. Coll. Yearbook, p. 1-42.

1942. Mykologiske Notitser II. Zent. Bakt. Paras. Infek. 104:409-412.

1945. Danish species of *Alternaria* and *Stemphylium*; Taxonomy, parasitism, economic significance. 560 pages. Copenhagen.

Neergaard, P., and Newhall, A. G.

1951. Notes on the physiology and pathogenicity of *Centrospora acerina* (Hartig) Newhall. Phytopath. 41:1021-1033.

Newhall, A. G.

1944. A serious storage rot caused by the fungus *Ansatospora macrospora* n. gen. Phytopath. 34:92-105.

1946. More on the name *Ansatospora acerina*. Phytopath. 36:893-894.

O'Leary, D. K.

1939. The utilization of cuticular compounds with reference to penetration by fungi. Thesis for degree of Ph.D., Cornell University. (Unpublished.)

Osterwalder, A.

1924. Über die durch *Cercospora macrospora* Osterwalder verursachte Blattkrankheit bei den Pensees. Mitt. Thurgau. Nat. Gesells. Heft. 25:59-80.

Pammel, L. H.

1895. Bacteriosis of rutabagas (*Bacillus campestris* n. sp.) Iowa Agr. Exp. Sta. Bul. 27:136.

Panassenko, V. T.

1930. Certain species of fungi of the Mucorales which cause storage rot. [In Czechoslovakian.] Sugar Indus. Sci. Notes. Kieff. 10:337-346. (Abstr. in Rev. Appl. Mycol. 5:590. 1926.)

Park, M.

1932. Report on the work of the mycological division. Ceylon Admin. Rept. Rept. of dir. of agr. for 1931: D-103 — D-111.

- Patel, M. K.
1929. Viability of certain plant pathogens in the soil. *Phytopath.* 19: 295-300.
- Paul, W. R. C.
1929. A comparative morphological and physiological study of a number of strains of *Botrytis cinerea* Pers. with special reference to their virulence. *Brit. Mycol. Soc. Trans.* 14:118-135.
- Peltier, G. L.
1915. Parasitic Rhizoctonias in America. *Ill. Agr. Exp. Sta. Bul.* 189: 283-390.
- Potter, M. C.
1899. White rot, a bacterial disease of the turnip. *Durham Phil. Soc. (England). Proc.* 1:165-167.
- Povah, A. H. W.
1917. A critical study of certain species of *Mucor* (historical and taxonomic.) *Torrey Bot. Club. Bul.* 44:241-259, 287-312.
- Pratt, O. A.
1918. Soil fungi in relation to disease of the Irish potato in southern Idaho. *J. Agr. Res.* 13:73-100.
- Rader, Wm. E.
1945. *Ansatospora acerina* found causing decay of carrots in Wayne County, New York. *Plant Dis. Repr.* 29:522. (Mimeo.)
1948. *Rhizoctonia carotae* n. sp. and *Gliocladium aureum* n. sp., two new root pathogens of carrots in cold storage. *Phytopath.* 30: 440-452.
- Rambousek, F.
1924. Berichte des Forschungs-Institutes der osl. Zucker-Industrie 1916. Die Rüben-Krankheiten in der Tschechoslowakei 1923. *Z. Zuckerind.* 69:197-201.
- Ramsey, G. B.
1925. *Sclerotinia* spp. causing decay of vegetables under transit and market conditions. *J. Agr. Res.* 31:597-632.
1934. Market pathology notes from Chicago. *Plant Dis. Repr.* 18: 40-41. (Mimeo.)
- Ramsey, G. B., and Smith, M. A.
1949. The occurrence of *Rhizoctonia crater rot* in Illinois-grown carrots. *Plant Dis. Repr.* 33:248-249.
- Remsberg, Ruth
1940. Studies on the genus *Typhula*. *Mycologia* 32:52-96.
- Richards, B. L.
1921. Pathogenicity of *Corticium vagum* on the potato as affected by soil temperatures. *J. Agr. Res.* 21:459-482.
- Rippel, A.
1940. Über die Verbreitung von *Aspergillus niger*. Insbesondere in Deutschland. *Arch. Mikrobiol.* 11:1-32.

Rolfs, F. M.

1902. Potato failures. Col. Agr. Exp. Sta. Bul. 91:1-33.

Salmon, E. S., and Ware, W. M.

1934. Report of Department of Mycology, Southeast. Agr. Coll., Wye, Kent. J. 33:17-27.

Scaramella, P.

1929. L'alternariosi o marciume nero della Carote. Boll. R. Staz. Pat. Veg. (n.s.) 9:226-237. (*Abstr. in Rev. Appl. Mycol.* 9:82. 1930.)

Schmidt, E. W.

1933. Zwei seltene Rübenschädlinge. Die Deutsche Zuckerind. 58: 17-18.

Shapovalov, M.

1919. Some potential parasites of the potato tuber. Phytopath. 9:36-42.

Smith, E. F.

1920. Jones' soft rot of carrots. In Bacterial diseases of plants, p. 223-252.

Snyder, W. C., and Hansen, H. N.

1940. The species concept in *Fusarium*. Amer. J. Bot. 27:64-67.

1945. The species concept in *Fusarium* with reference to discolor and other sections. Amer. J. Bot. 32:657-777.

Strohbinder, M. F., and Driabina, Mme. M. M.

1935. An experiment on the use of the Conn-cholodny method for the microbiological investigation of vegetables and fruits during storage. [In Russian.] Microbial. 4:379-384.

Thom, C., and Raper, K. B.

1945. A manual of the Aspergilli. 373 pages.

Tisdale, W. B., and Kelley, J. B.

1926. A phytophthora disease of tobacco. Fla. Agr. Exp. Sta. Bul. 179:159-219.

Tompkins, C. M., and Hansen, H. N.

1950. Pansy leafspot, caused by *Centrospora acerina*, host range and control. Hilgardia 19:383-398.

Tompkins, C. M., Richards, B. L., Tucker, C. M., and Gardner, W. M.

1936. Phytophthora root rot of sugar beet. J. Agr. Res. 52:205-216.

Truscott, J. H. L.

1944. A storage rot of celery caused by *Ansatospora macrospora* (Osterw.) Newhall. Canad. J. Res. C. 22:290-304.

Tucker, C. M.

1933. The distribution of the genus *Phytophthora*. Mo. Res. Bul. 184:3-80.

Vasudeva, R. Sahai.

1930. Studies in the physiology of parasitism. XI. An analysis of the factors underlying specialization of parasitism, with special reference to the fungi *Botrytis allii* Munn and *Monilia fructigena* Pers. Ann. Bot. 44:469-493.

- Virgin, W. J.
1942. Investigations of storage diseases of carrots. (*Abstr.*) *Phytopath.* 32:830.
- Waterston, J. M.
1938. Report of the plant pathologist, 1937. *Bd. Agr. Bermuda. Rept.* 1937:24-37.
- Westerdijk, Johanna, and van Luijk, A.
1924. Eine Anthraknose des Kümmels (*Carum carvi*) Meded. *Phytopath.* 8:51-54.
- White, N. H.
1945. Fungal soft rot of carrots. *Tasmanian J. Agr.* 16:59-60.
- White, R. P.
1926. *Rhizoctonia* crown rot of carrots. *Phytopath.* 16:367-368.
- Wiant, J. S., and Tucker, C. M.
1940. A rot of winter queen watermelons caused by *Phytophthora capsici*. *J. Agr. Res.* 9:73-88.
- Williams, P. H., Ainsworth, G. C., Oyler, E., White, H. L., and Reed, W. H.
1938. Plant diseases. *Exp. Res. Sta. Cheshunt. Rept.* 1937:42-58.
- Wollenweber, H. W., and Reinking, A. O.
1935. Die Fusarien-ihre Beschreibung, Schadwirkung und Bekämpfung. 355 pages. Berlin.
- Young, P. A., and Morris, H. E.
1927. Sclerotinia wilt of sunflowers. *Mont. Agr. Exp. Sta. Bul.* 208:1-32.

Appendix

A Key to Diseases of Stored Carrots

- I. Rot soft, watery, usually slimy with no evidence of mold growth, sometimes with foul odor.
 - Bacterial soft rot—Erwinia carotovora* p. 7
- II. Decay not slimy, cells if separating accompanied by obvious mold growth.
 - A. Sclerotia present
 1. Hyphae absent from surface of lesion, sclerotia in lesion round resembling radish seeds.
 - Buckshot rot—Typhula* spp. p. 48
 2. Surface hyphae usually present, sclerotia irregular in shape and usually large.
 - a. Sclerotia covered with dark to black rind, but white inside.
 - *Sclerotia very contorted, closely appressed to host, conidia usually present, decay firm.
 - Grey mold rot—Botrytis cinerea* p. 14
 - **White mycelium of pathogen present, conidia absent, sclerotia embedded in mycelium, decay soft and watery, slightly discolored, and separating easily from healthy tissue.
 - Cottony soft rot—Sclerotinia sclerotiorum* p. 10
- b. Sclerotial rind absent, homogenous throughout, usually brown.
 - *Hyphae brown, lesions not sunken and usually on the crown.

- Brown crown rot—Pellicularia filamentosa*..... p. 33
- **Hyphae usually white, lesions anywhere on root, sunken, and usually lined with white hyphae, sclerotia when present, minute, light to medium brown.
- Crater rot—Rhizoctonia carotae*..... p. 19
- B. Sclerotia absent.
1. Decay black or very dark brown.
 - a. Advancing margins black and sharply delimited.

Black rot—Stemphylium radicinum..... p. 26
 - b. Advancing margins brown and less distinct.

Licorice rot—Centrospora acerina..... p. 24
 2. Decay not black.
 - a. Localized into sunken cankers on side of root.

*Surface covered with sooty black conidia of fungus, decay brown, very shallow and leathery.

Sooty rot—Aspergillus niger..... p. 45

**Surface covered with loose white cottony mycelium, decay firm but not punky, slightly discolored.

Crater rot—Rhizoctonia carotae..... p. 19

***Surface not covered with hyphae, or if present, light tan, pinkish or green in color.

†Lesion light brown color, shallow (1 mm), very hard and leathery, and hyphae, if present, tan or pinkish.

Hard rot—Gliocladium aureum..... p. 47

††Lesion not discolored.

‡Decay under lesion dry and punky, though in younger stages indistinguishable from Gliocladium hard rot.

Dry rot—Fusarium roseum..... p. 31

‡‡Decay more leathery, sometimes greyish or greenish colored.

Blue-green mold rot—Penicillium expansum..... p. 43
 - b. Decay not localized in cankers on side of root, but involving crown or entire root.

*Decay chiefly of the crown, brownish, tissues soft, mushy but not watery.

Brown crown rot—Pellicularia filamentosa..... p. 33

**Decay progressive, involving entire root.

†Diseased tissues soft, watery, easily separating, only a slight discoloration, usually covered with white mycelial growth.

Cottony soft rot—Sclerotinia sclerotiorum..... p. 10

††Tissues not sharply delimited and separable.

‡Roots seldom covered with mycelial growth or if present only sparse in advanced stages.

§Decay brown from start, turning greyish, very watery but rarely separating.

Slate rot—Pythium..... p. 38

§§Decay at first only water-soaked, turning darker, wet but firm and never greyish (indistinguishable from Pythium in early stages).

Rubbery brown rot—Phytophthora..... p. 35

‡‡Roots usually covered with luxuriant mycelial growth.

§Growth white at first, turning blue or green.

Blue-green mold rot—Penicillium expansum..... p. 43

§§Growth some shade of brown or grey.

¶Diseased tissue light brown, spongy to almost leathery in advanced stages, rarely a wet rot, hyphae usually rather fine.

Grey mold rot—Botrytis cinerea..... p. 14

¶¶Tissues scarcely discolored, water-soaked wet rot, hyphae very large and coarse.

||Hyphae bristlelike, usually brown or grey, spores borne on single stalks, common at lower temperatures.

Whisker rot—Mucor spp...... p. 40

|||Hyphae more tangled, greyish to blackish, spores borne on stalks which are joined at bases in clusters, more common at higher temperatures.

Woolly rot—Rhizopus arrhizus..... p. 30